“A CLINICAL STUDY ON PROGNOSTIC FACTORS IN DUODENAL ULCER PERFORATION”

A DISSERTATION SUBMITTED TO THE TAMILNADU
Dr. MGR MEDICAL UNIVERSITY

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In partial fulfilment of the Regulations

for the award of the Degree of

M.S. (GENERAL SURGERY) BRANCH-I

DEPARTMENT OF GENERAL SURGERY

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI

MAY 2018

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Lastly, I express my thanks to my patients without whom this study would not have been possible.

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PROTOCOL TITLE: A CLINICAL STUDY ON PROGNOSTIC FACTORS IN DUODENTAL ULCER PERFORATION.

PRINCIPAL INVESTIGATOR: DR.A.RAMPRASATH, MBBS.,

DESIGNATION OF PRINCIPAL INVESTIGATOR POST GRADUATE IN GENERAL SURGRY
DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE , TIRUNELVELI

Dear , Dr.A.Ramprasath, MBBS., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 10.12.15.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Biocthics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occured in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
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   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

Dr.K.Shantaraman MD
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CERTIFICATE - II

This is certify that this dissertation work title **"A CLINICAL STUDY ON PROGNOSTIC FACTORS IN DUODENAL ULCER PERFORATION"** of the candidate **Dr.A.RAMPRASATH** with registration Number **221511356** for the award of **M.S.** in the branch of **GENERAL SURGERY.** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **0 PERCENTAGE** of plagiarism in the dissertation.

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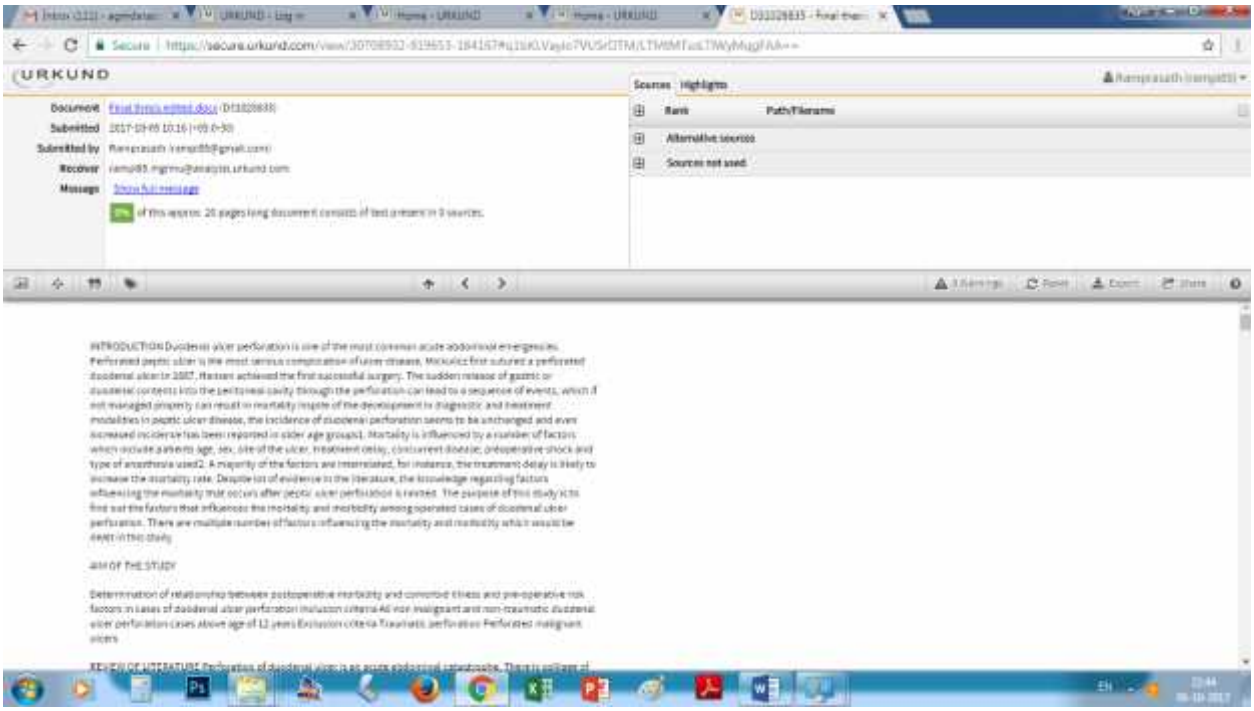
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INTRODUCTION Duodenal ulcer perforation is one of the most common acute abdominal emergencies.
AIM OF THE STUDY
Determination of relationship between postoperative morbidity and comorbid illness and pre-operative risk factors in cases of duodenal ulcer perforation Inclusion criteria All non malignant and non traumatic duodenal ulcer perforation cases above age of 12 years Exclusion criteria Traumatic perforation Perforated malignant ulcers

INTRODUCTION

Duodenal ulcer perforation is one of the most common acute abdominal emergencies. Perforated peptic ulcer is the most serious complication of ulcer disease, Mickulicz first sutured a perforated duodenal ulcer in 1887, Hansen achieved the first successful surgery.

The sudden release of gastric or duodenal contents into the peritoneal cavity through the perforation can lead to a sequence of events, which if not managed properly can result in mortality

Inspite of the development in diagnostic and treatment modalities in peptic ulcer disease, the incidence of duodenal perforation seems to be unchanged and even increased incidence has been reported in older age groups[1].

Mortality is influenced by a number of factors which include patients age, sex, site of the ulcer, treatment delay, concurrent disease, preoperative shock and type of anasthesia used[2]. A majority of the factors are interrelated, for instance, the treatment delay is likely to increase the mortality rate.

Despite lot of evidence in the literature, the knowledge regarding factors influencing the mortality that occurs after peptic ulcer perforation is limited.

The purpose of this study is to find out the factors that influences the mortality and morbidity among operated cases of duodenal ulcer perforation. There are multiple number of factors influencing the mortality and morbidity which would be dealt in this study.

AIM OF THE STUDY

Determination of relationship between postoperative morbidity and comorbid illness and pre-operative risk factors in cases of duodenal ulcer perforation

Inclusion criteria

All non malignant and non-traumatic duodenal ulcer perforation cases above age of 12 years

Exclusion criteria

Traumatic perforation

Perforated malignant ulcers

REVIEW OF LITERATURE

Perforation of duodenal ulcer is an acute abdominal catastrophe.

There is spillage of intestinal contents into the peritoneal cavity resulting in peritonitis, fluid and electrolyte imbalance, circulatory insufficiency, septicemia and finally death.

DUODENAL ULCER PERFORATION

Incidence:

The incidence of perforated peptic ulcer is approximately 7 to 10 cases per 1000000 populations per year.

Perforation is found in about 7% of patients hospitalized for peptic ulcer disease and is the first manifestation of the disease in about 2% of patients with duodenal ulcer. It is estimated that, after the diagnosis of duodenal ulcer,0.3% of patients perforate annually in the first 10 years. Whether eradication of HP will reduce this incidence is as yet unknown.

In the duodenum, the ulcers that perforate are located anteriorly, and the aphorism that “anterior ulcers perforate, posterior ones bleed” is as relevant today as ever.

Epidemiology:

There has been a considerable change in the epidemiology of perforated peptic ulcer over the last two decades.

ANATOMY OF THE DUODENUM

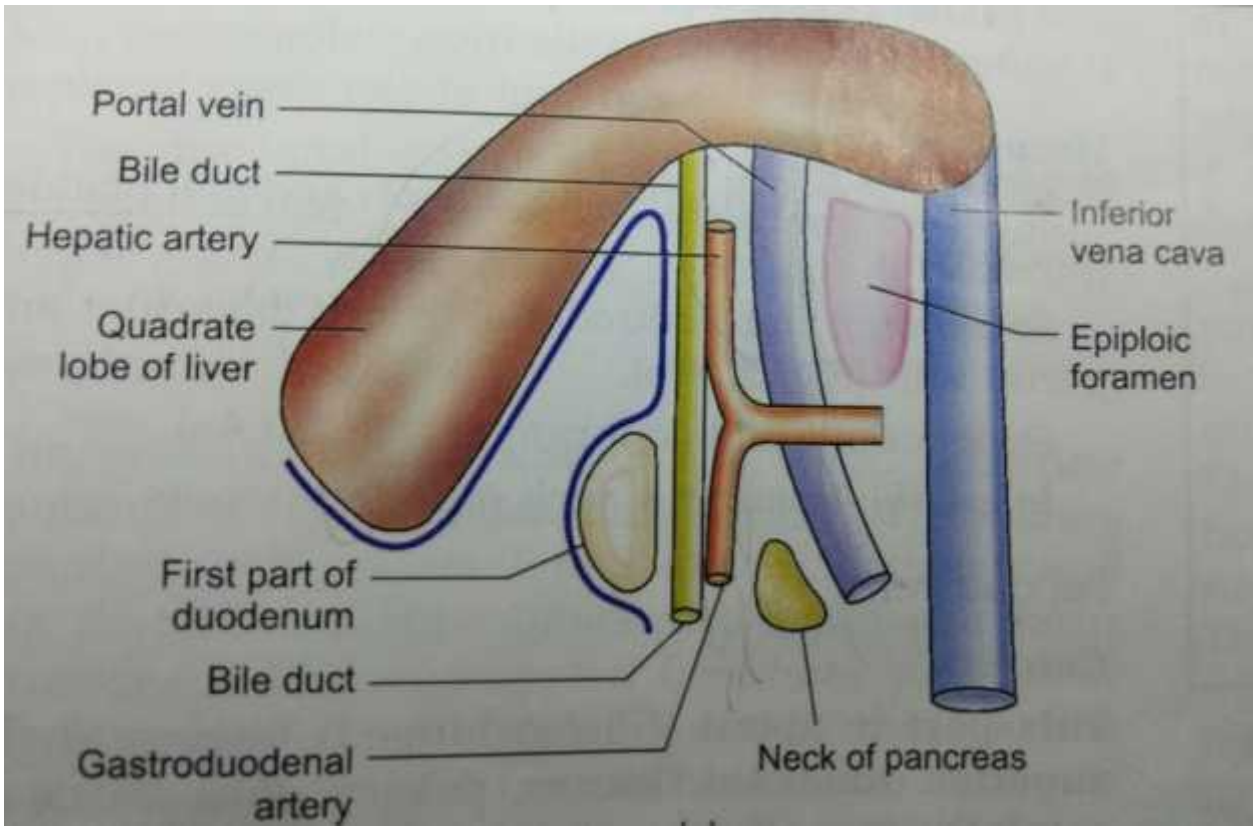
The entire is duodenum is about 20 to 25 cm long and is the widest, shortest and most sessile part of the small intestine. It is devoid of mesentry and is only partially covered by peritoneum and constantly curved incomplete circle encircling the head of the pancreas.

It is situated entirely above the umbilicus starting from the pylorus it pass backwards up and extend to the right for 5cms inferior to the posterior part of the quadrate lobe of liver to the neck of the gallbladder. Its direction varies according to the distension of the stomach, it then curves abrubtly to 7.5cms to the medial part of right kidney, usually upto the level of 3rd lumbar vertebral body. At its 2nd bend it turns horizontally left across the vertebral column for 5 to 10 cms, just above the level of umbilicus with a slight curve upwards, it ascends then infront and to the left of aorta for 2.5cms, ending opposite to the 2nd lumbar vertebra in the jejeunum. At this junction it turns abrubtly forwards, in the DJ flexure which is about 2.5cms left of the midline and one cm below the transpyloric plane. For

descriptive purpose the duodenum is divided into superior , descending , horizontal and ascending parts.

Relationship of duodenum:

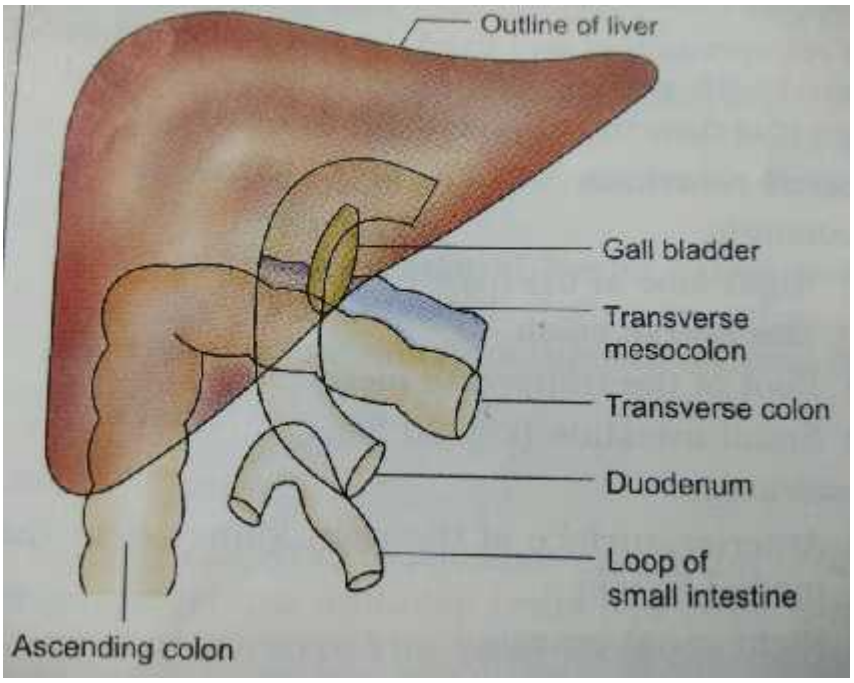
First part:

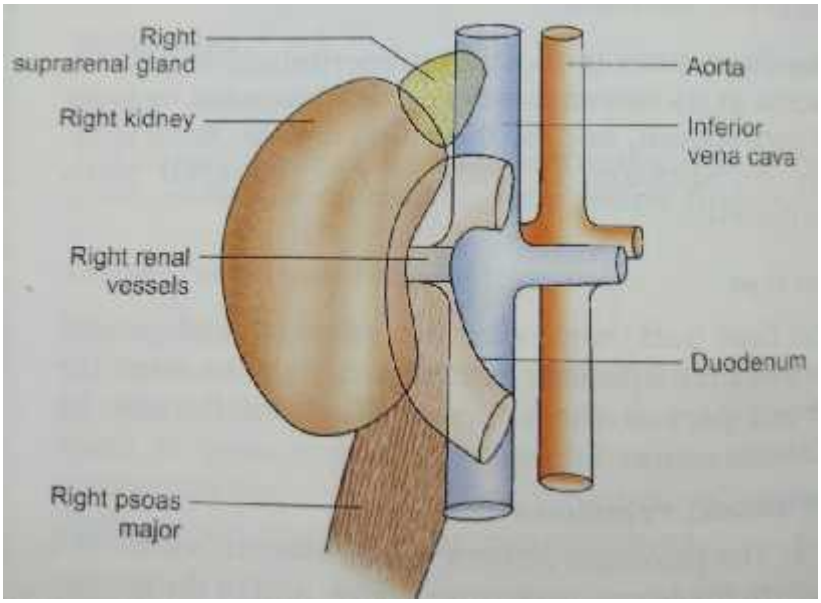


It is about 5cm long and is the most mobile part of the duodenum, it extends from the pylorus of the stomach to the gallbladder neck. It is covered by peritoneum in its anterior aspect but the posterior part is devoid of peritoneum except near the pylorus for 2.5cms. it is related above to the quadrate lobe of the

liver and posteriorly with the epiploic foramen, behind by the gastroduodenal artery, bileduct and portalvein, and posteroinferiorly by the head of the pancreas.

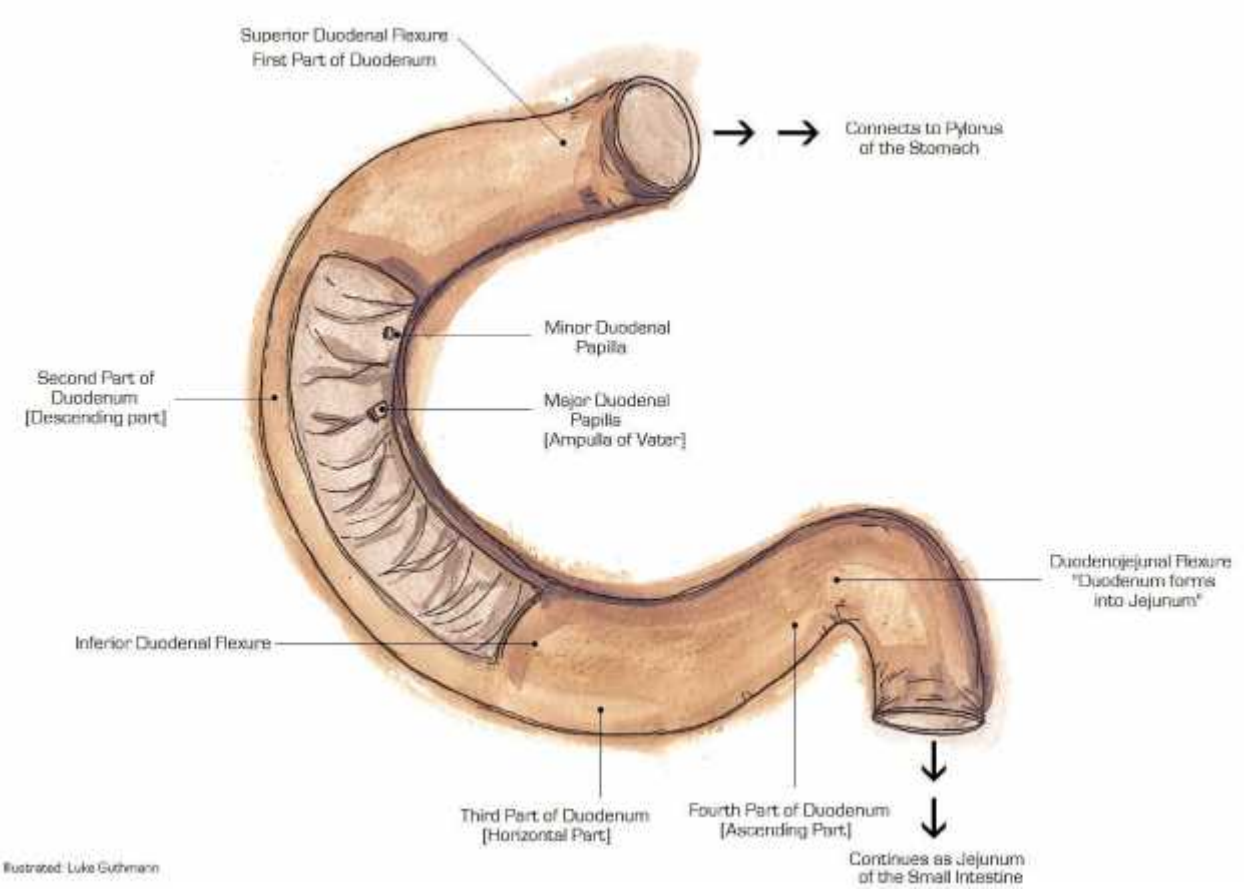
Second part:





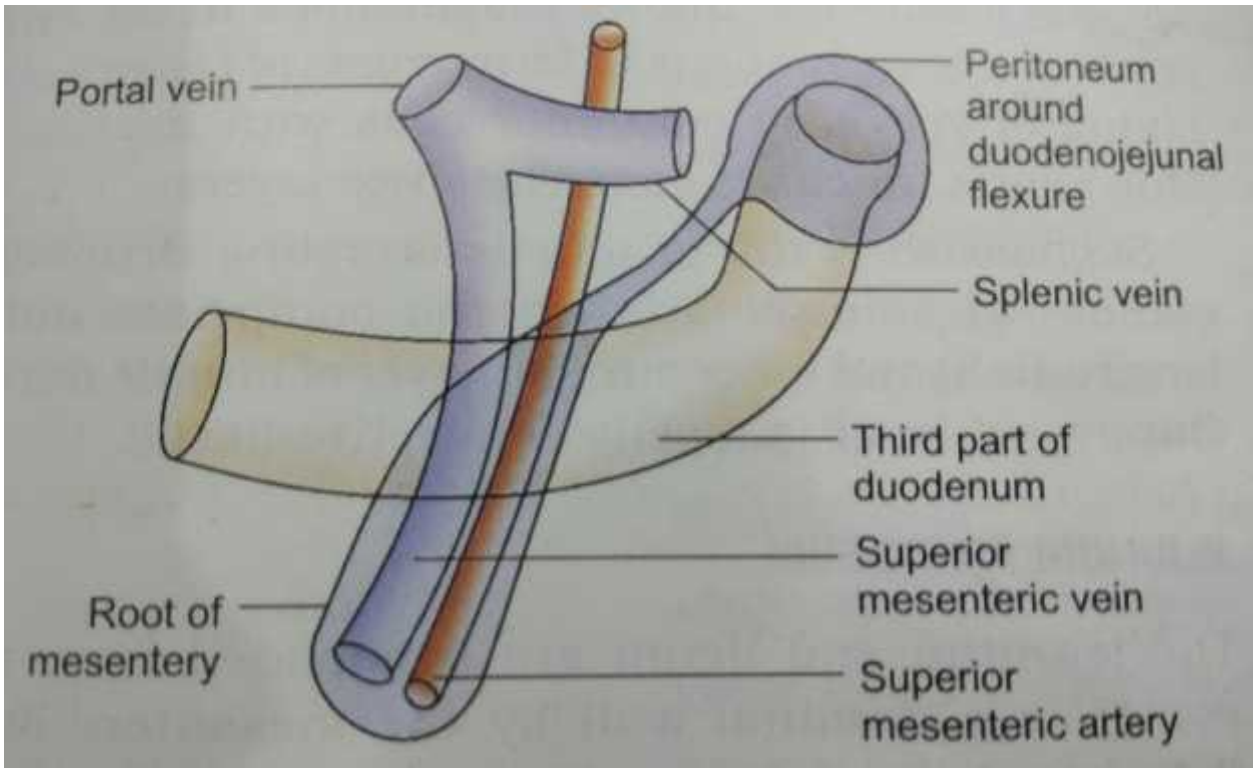
It descends from the neck of the gall bladder and is about 8 to 10cms long to the lower border of the 3rd lumbar vertebral body crossed by the transverse colon. It is related infront to the right lobe of the liver, transverse colon and jejunum.

It is related behind to the right kidney and connected to right kidney by loose connective tissue, medial relationship include head of the pancreas and bile duct. Lateral relationship include the right colic flexure. A bit of pancreatic head is embedded on the duodenal wall. The pancreatic and bile duct has contact in its medial side where it unites to form the hepatopancreatic ampulla. The distal narrow end of this opens in the major duodenal papilla which is situated posteromedially in the descending duodenum which inturn is situated 8 to 10cm distal to the pylorus.



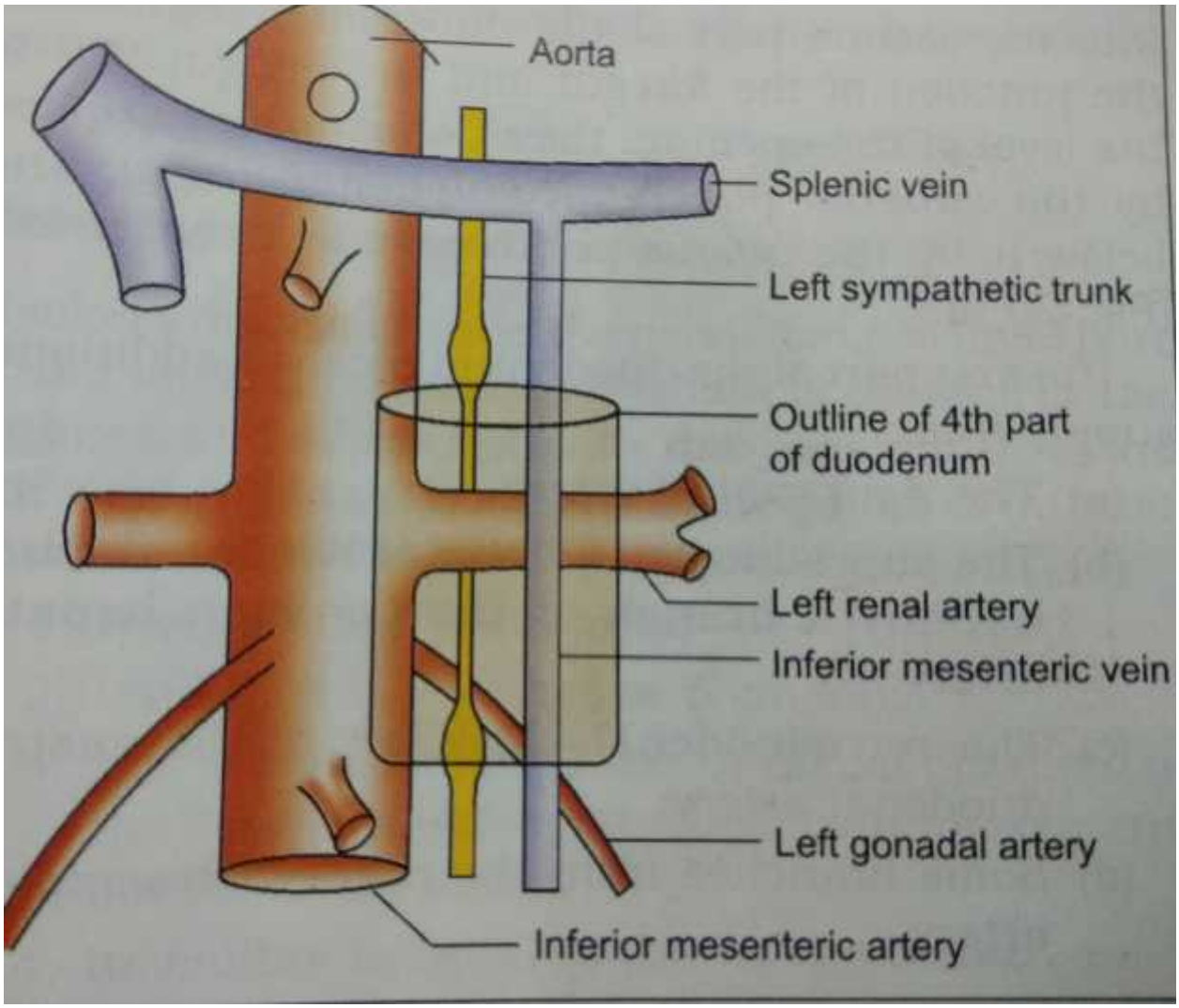
There may be an accessory pancreatic duct opening 2cm above the major duodenal papilla which is the minor duodenal papilla.

Third part:



It is about 10cm long and it extends from the lower border of 3rd lumbar vertebra to end in the fourth part infront of the abdominal aorta . The anterior surface of this part is crossed with peritoneum, except in the midline where it is crossed by the superior mesenteric vessels and root of the mesentry. The posterior surface is covered by peritoneum at its left end. Posterior surface is formed by right ureter, right psoas major, right gonodal vessels, inferior vena cava and abdominal aorta.

Fourth part:



This part is 2.5 cm long and ascends to the left of aorta to the upper border of 2nd lumbar vertebra ending in the duodenojejunal flexure. Posterior relations include left sympathetic trunk, left psoas major, left renal and gonadal vessels, to its left

are left kidney and left ureter, above by the head of the pancreas and infront by the transverse colon and transverse mesocolon.

PERITONEAL ATTACHMENTS:

The first part of the duodenum is slightly mobile and the other parts are fixed.

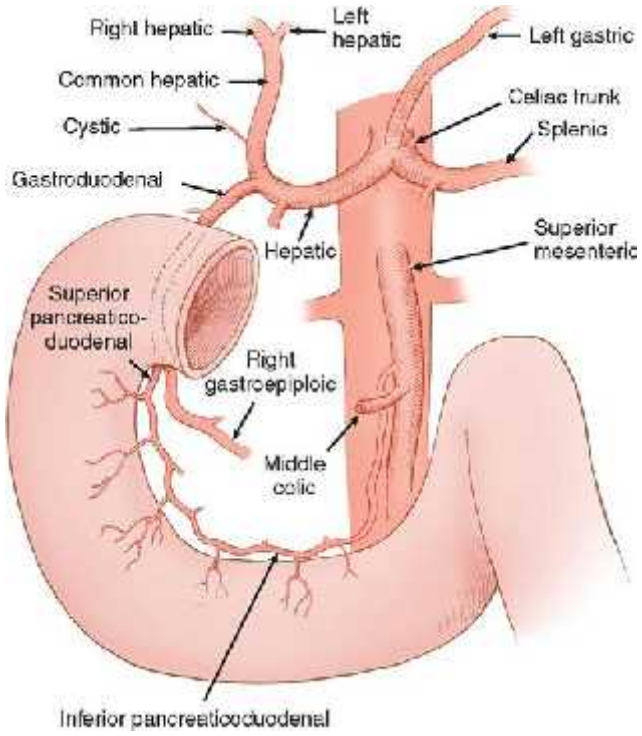
The duodenojejunal flexure is positioned by the ligament of treitz or suspensory muscle and consists of 2 parts

A slip of skeletal muscle from the diaphragm near the oesophageal opening ending in a connective tissue near the celiac artery

A fibromuscular band of smooth muscle from the 3rd and fourth part of the duodenum to the same pericoeliac connective tissue.

BLOOD SUPPLY OF THE DUODENUM

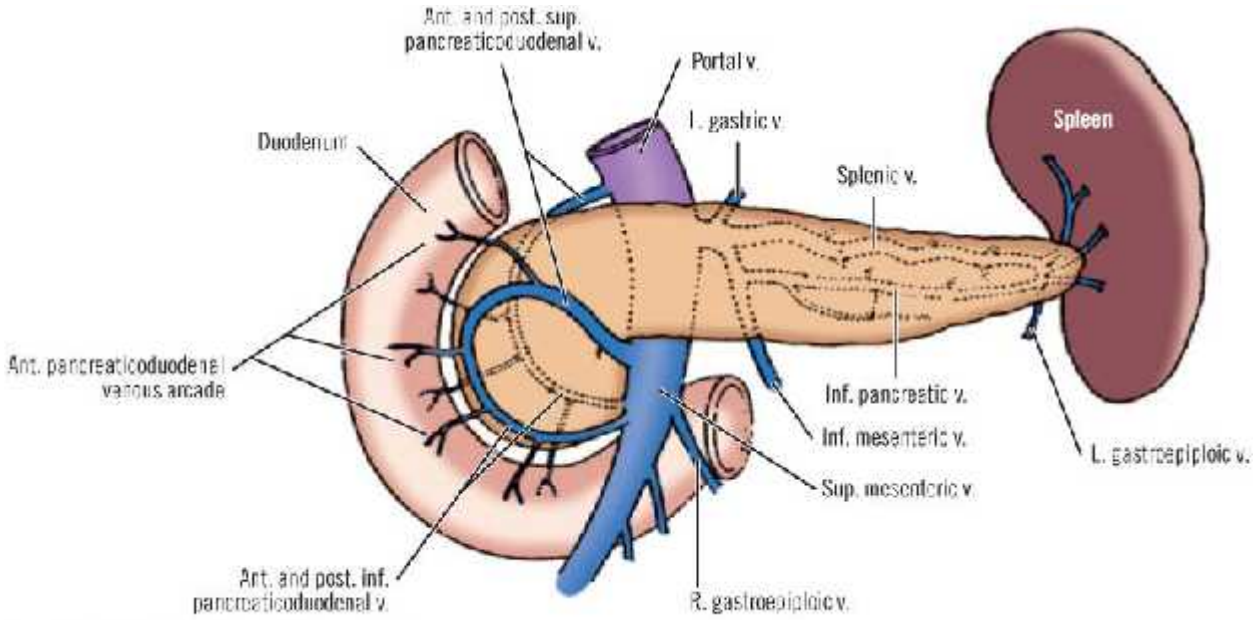
ARTERIAL SUPPLY:



It is supplied by the supraduodenal, right gastro epiploic artery and superior and inferior pancreaticoduodenal arteries. The first part of the duodenum also receives branches from the hepatic artery proper and gastroduodenal artery, these branches also supply the pyloric canal

VENOUS SUPPLY:

The veins end in the splenic, superior mesenteric and portal veins



NERVE SUPPLY:

They are supplied from the celiac plexus.

HISTOLOGY:

The wall of the duodenum consist of 4 layers

1. Mucosa
2. Submucosa
3. Muscularispropria
4. Serosa

The mucosa is the innermost layer and consists of three layers

- Epithelium
- Lamina propria
- Muscularis mucosa

The epithelium is exposed to the intestinal lumen and through this absorbtion and secretion occurs.

The lamina propria is located immediately external to the epithelium and consists of connective tissue and hetergenous population of cells, it is demarcated by the thin sheet of smooth muscle cells from the muscularis mucosa.

The mucosa is organized into villi and crypts. Villi are finger like projections of epithelium and underlying laminapropria that contain blood and lymphatic vessels that extend into the intestinal lumen

The submucosa consists of dense connective tissue and heterogenous population of cells including leucocytes and fibroblasts.

The muscularis propria consists of an outer longitudinal oriented layer and an inner circularly oriented smooth muscle fibre.

The serosa consists of single layer of mesothelial cells and it is a component of visceral peritoneum.

MICROSCOPIC ANATOMY OF STOMACH AND DUODENUM

The gastric epithelial cells are mucus producing and are turned over rapidly. In the pyloric part of the stomach, and also the duodenum, mucus-secreting glands are seen. Most of the special cells of the stomach (parietal and chief cells) are found in the gastric crypts .

The stomach has also numerous endocrine cells.

PARIETAL CELLS

These are seen in the body (acid-secreting portion) of the stomach and line the gastric crypts, being more abundant distally. They are responsible for the production of hydrogen ions to form hydrochloric acid. The hydrogen ions are actively secreted by the proton pump, a hydrogen–potassium-ATPase, which exchanges intraluminal potassium for hydrogen ions. The potassium ions enter the lumen of the crypts passively, but the hydrogen ions are pumped against an immense concentration gradient (1 000 000:1).

CHIEF CELLS

These lie principally proximally in the gastric crypts and produce pepsinogen. Two forms of pepsinogen are described: pepsinogen I and pepsinogen II. Both are produced by the chief cell, but pepsinogen I is produced only in the

stomach. The ratio between pepsinogens I and II in the serum decreases with gastric atrophy. Pepsinogen is activated in the stomach to produce the digestive protease, pepsin. Endocrine cells:

The stomach has numerous endocrine cells, which are critical to its function. In the gastric antrum, the mucosa contains G cells, which produce gastrin. Throughout the body of the stomach, enterochromafn-like (ECL) cells are abundant and produce histamine, a key factor in driving gastric acid secretion. In addition, there are large numbers of somatostatin-producing D cells throughout the stomach, and somatostatin has a negative regulatory role.

DUODENUM

The duodenum is lined by a mucus-secreting columnar epithelium. In addition, Brunner's glands lie beneath the mucosa and are similar to the pyloric glands in the pyloric part of the stomach. Endocrine cells in the duodenum produce cholecystokinin and secretin.

PHYSIOLOGY OF STOMACH AND DUODENUM:

The stomach mechanically breaks up ingested food particles and together by the action of acid and pepsin, forms chime that passes into the duodenum, in contrast to the acidic environment of the stomach, the duodenal environment is alkaline.

It is due to the presence of secretion of bicarbonate ions from both the pancreas and the duodenum. This neutralizes the acid chime and adjusts the luminal osmalarity to approximate to that of plasma.

Endocrine cells in the duodenum produce cholecystokinin, which stimulates the pancreas to produce trypsin and the gallbladder to contract. Secretin is also produced by the endocrine cells of the duodenum, this hormone inhibits the gastric acid secretion and promotes the production of bicarbonate by pancreas

GASTRIC MUCUS AND THE GASTRIC MUCOSAL BARRIER:

The gastric mucous layer is essential to the integrity of the gastric mucosa. It is a viscid layer of mucopolysaccharides produced by the mucus-producing cells of the stomach and the pyloric glands.

Gastric mucus is an important physiological barrier to protect the gastric mucosa from mechanical damage, and also the effects of acid and pepsin. Its considerable buffering capacity is enhanced by the presence of bicarbonate ions within the mucus.

Many factors can lead to the breakdown of this gastric mucous barrier. These include bile, non-steroidal anti-inflammatory drugs (NSAIDs), alcohol, trauma and shock.

Tonometry studies have shown that, of all the gastrointestinal tract, the stomach is the most sensitive to ischemia following a hypovolemic insult and also the slowest to recover. This may explain the high incidence of stress ulceration in the stomach.

PEPTIDES AND NEUROPEPTIDES IN THE STOMACH AND DUODENUM:

As with most of the gastrointestinal tract, the endocrine cells of the stomach produce peptide hormones and neurotransmitters. Previously, nerves and endocrine cells were considered distinct in terms of their products. However, it is increasingly realised that there is enormous overlap within these systems. Many peptides recognised as hormones may also be produced by neurones, hence the term neuropeptides. The term ‘messenger’ can be used to describe all such products.

There are three conventional modes of action that overlap

1. Endocrine. The messenger is secreted into the circulation, where it affects tissues that may be remote from the site of origin

2 .Paracrine. Messengers are produced locally and have local effects on tissues. Neurones and endocrine cells both act in this way.

3 Neurocrine (classical neurotransmitter).

Messengers are produced by the neurone via the synaptic knob and pass across the synaptic cleft to the target. Many peptide hormones act on the intrinsic nerve plexus of the gut and influence motility. Similarly, neuropeptides may influence the structure and function of the mucosa.

GASTRODUODENAL MOTOR ACTIVITY:

The motility of the entire gastrointestinal tract is modulated to a large degree by its intrinsic nervous system.

Critical in this process is the migrating motor complex (MMC). In the fasting state, and after food has cleared, in the small bowel there is a period of quiescence lasting for a period of 40 minutes (phase I). There follows a series of waves of electrical and motor activity, lasting for about 40 minutes, propagated from the fundus of the stomach in a caudal direction at a rate of about 3 per minute (phase II). These pass as far the pylorus, but not beyond. Duodenal slow waves are generated in the duodenum at a rate of about 10 per minute, which carry down the small bowel. The amplitude of these contractions increases to a maximum in phase III, which lasts for about 10 minutes.

This 90-minute cycle activity is then repeated. From the duodenum, the MMC moves distally at 5–10 cm/min, reaching the terminal ileum after 1.5 hours.

Following a meal, the stomach exhibits receptive relaxation, which lasts for a few seconds. Following this, adaptive relaxation occurs, which allows the proximal stomach to act as a reservoir. Most of the peristaltic activity is found in the distal stomach (the antral mill) and the proximal stomach demonstrates only tonic activity.

The pylorus, which is most commonly open, contracts with the peristaltic wave and allows only a few millilitres of chyme through at a time. The antral contraction against the closed sphincter is important in the milling activity of the stomach. Although the duodenum is capable of generating ten waves per minute, after a meal it only contracts after an antral wave reaches the pylorus. The coordination of the motility of the antrum, pylorus and duodenum means that only small quantities of food reach the small bowel at a time.

Motility is influenced by numerous factors, including mechanical stimulation and neuronal and endocrine influences.

INVESTIGATION OF THE STOMACH AND DUODENUM

FLEXIBLE ENDOSCOPY

Flexible endoscopy is the ‘gold standard’ investigation of the upper gastrointestinal tract. Flexible endoscopy is more sensitive than conventional radiology in the assessment of the majority of gastroduodenal conditions A video-

gastroscope. (a) The camera stack. (b) The gastroscope, and biopsy forceps, in the working channel of endoscopic therapy. Fibreoptic endoscopy is generally a safe investigation, but it is important that all personnel undertaking these procedures are adequately trained and that resuscitation facilities are always available. A more experienced endoscopist will have a higher index of suspicion for any mucosal abnormalities and will take more biopsies. Spraying the mucosa with dye endoscopically may allow better discrimination between normal and abnormal mucosa, so allowing a small cancer to be more easily seen. In the future, advances in technology may allow 'optical biopsy' to determine the nature of mucosal abnormalities in real time. Upper gastrointestinal endoscopy can be performed without sedation, but when sedation is required incremental doses of a benzodiazepine are usually administered. Sedation is of particular concern in the case of gastrointestinal bleeding as it may have a more profound effect on the patient's cardiovascular stability. It has now become the standard to use pulse oximetry to monitor patients during upper gastrointestinal endoscopy, and nasal oxygen is often also administered. Buscopan is useful to abolish duodenal motility for examinations of the second and third parts of the duodenum. Examinations of this type are best carried out using a side-viewing endoscope such as is used for endoscopic retrograde cholangiopancreatography (ERCP). Some patients are relatively resistant to sedation with benzodiazepenes, particularly those who are

accustomed to drinking alcohol. Increasing the dose of benzodiazepines in these patients may not result in any useful sedation, but merely make the patient more restless and confused. Such patients are sometimes better endoscoped fully awake using a local anaesthetic throat spray and a narrow-gauge endoscope. Whatever the circumstances, it is important that resuscitation facilities are available, including agents that reverse the effects of benzodiazepines, such as umazenil. The technology associated with upper gastrointestinal endoscopy is continuing to advance. Instruments which allow both endoscopy and endoluminal ultrasound to be performed simultaneously are used routinely. Bleeding from the stomach and duodenum can be treated with a number of haemostatic measures. These include injection with various substances, diathermy, heater probes and lasers. These approaches appear to be useful in the treatment of bleeding ulcers, although there are few good controlled trials in this area. There is no good evidence that such interventional procedures at the moment work in patients who are bleeding from very large vessels, such as the gastroduodenal artery or splenic artery, although technology may overcome this problem in the future.

UPPER GASTROINTESTINAL RADIOLOGY

It is not used as much as in previous years, as endoscopy is a more sensitive investigation for most gastric problems. Computed tomography (CT) imaging with oral contrast has also replaced contrast radiology in many of the areas where

anatomical information is sought, e.g. large hiatus hernias of the rolling type and chronic gastric volvulus. In these conditions it may be difficult for the endoscopist to determine exactly the anatomy or, indeed, negotiate the deformity to see the distal stomach.

ULTRASONOGRAPHY

Standard ultrasound imaging can be used to investigate the stomach, but used conventionally it is less sensitive than other modalities. In contrast, endoluminal ultrasound and laparoscopic ultrasound are probably the most sensitive techniques available in the preoperative staging of gastric cancer. In endoluminal ultrasound, the transducer is usually attached to the distal tip of the instrument. However, devices have been developed which may be passed down the biopsy channel, albeit with poorer image quality. Five layers of the gastric wall may be identified on endoluminal ultrasound and the depth of invasion of a tumour can be assessed with exquisite accuracy. Enlarged lymph nodes can also be identified and the technique's accuracy in this situation is about 80 per cent. Finally, it may be possible to identify liver metastases not seen on axial imaging. Laparoscopic ultrasound is also a very sensitive imaging modality to a large measure because of the laparoscopy itself . It is one of the most sensitive methods of detecting liver metastases from gastric cancer. An additional use of ultrasound is in the assessment of gastric emptying. Swallowed contrast is utilised, which is

designed to be easily seen using an ultrasound transducer. The emptying of this contrast is then followed directly. The accuracy of the technique is similar to that of radioisotope gastric emptying studies .

CT SCANNING AND MAGNETIC RESONANCE IMAGING

The resolution of CT scanners continues to improve, and multislice CT is of increasing value in the investigation of the stomach, especially in gastric malignancies . The presence of gastric wall thickening associated with a carcinoma of any reasonable size can be easily detected by CT, but it lacks sensitivity in detecting smaller and curable lesions. It is much less accurate in 'T' staging than endoluminal ultrasound. Lymph node enlargement can be detected and, based on the size and shape of the nodes, it is possible to be reasonably accurate in detecting nodal involvement with tumour. However with all imaging techniques, it is limited. Microscopic tumour deposits in lymph nodes cannot be detected when node is not enlarged and, in contrast, lymph nodes may undergo reactive enlargement but may not contain tumour. These problems apply to all imaging techniques. The detection of small liver metastases is on the rise , although in general terms metastases from gastric cancer are less easy to detect using CT than those, for instance, from colorectal cancer. This is because metastases from gastric cancer may be of the same density as liver and may not handle the intravenous

contrast any differently. At present, magnetic resonance imaging (MRI) scanning does not offer any specific advantage in assessing the stomach

LAPAROSCOPY

This technique is now well used in the assessment of patients with gastric cancer. Its particular value is in the detection of peritoneal disease, which is difficult by any other technique, unless the patient has ascites or bulky intraperitoneal disease. Its main limitation is in the evaluation of posterior extension but other techniques are available to evaluate posterior invasion, especially CT and endoluminal ultrasound. Usually laparoscopy is combined with peritoneal cytology unless laparotomy follows immediately.

GASTRIC EMPTYING STUDIES

These are useful in the study of gastric dysmotility problems, particularly those that follow gastric surgery. The principle of the examination is that a radioisotope-labelled liquid and solid meal are ingested by the patient and the emptying of the stomach is followed on a gamma camera. This allows the proportion of activity in the remaining stomach to be assessed numerically, and it is possible to follow liquid and solid gastric emptying independently

RISK FACTORS OF PERFORATION :

Age:

Previously more common in the middle aged between 30 to 50 years. Increasing use of NSAID has resulted in an increased incidence in 6th and 7th decade.

Sex:

Previously male to female ratio is 2:1, now perforation occurs most commonly in elderly female patients.

Occupation:

More common in lower socioeconomic status.

SEASON:

More common in winter season.

Drug intake:

A strong association has been observed between the use of non steroidal anti-inflammatory agents (NSAIDs) and perforation of duodenal ulcers. The use of these drugs appears to be the major precipitating factor in currently treated patients[3]. A second risk factor for perforation is immune suppression, particularly

among transplant patients treated with steroids. Other factors include increasing patient age, chronic obstructive lung disease, major burns and multiple organ system failure.

PATHOPHYSIOLOGY:

Ultimately, peptic ulceration results when the caustic effects of acid and pepsin in the gastrointestinal lumen overwhelm the ability of the mucosa to resist those effects. The gastroduodenal mucosa is exposed to acid and pepsin continuously, yet ulceration is an abnormal event. The mechanisms that normally enable the mucosa to resist acid-peptic attack that can be divided into three major components :pre-epithelial, epithelial and postepithelial defence mechanisms.

PRE-EPITHELIAL DEFENCE MECHANISMS:

The pre-epithelial defense mechanisms are features that impede contact between epithelial cells and noxious agents in the gastrointestinal lumen. Gastric and duodenal epithelial cells normally are shielded from acid-peptic attack by a prominent coat of mucus and by a layer of unstirred water that is rich in bicarbonate[4]. Both mucus and bicarbonate are secreted into the lumen by gastric epithelial cells and by Brunner's glands in the duodenum. Bicarbonate from the blood also enters the unstirred water layer through the process of paracellular diffusion. Within the mucus layer, glycoproteins form a physical barrier to the

diffusion of pepsin, and the bicarbonate ions that accompany the glycoproteins can neutralize acid. Mucus also contains substantial quantities of surface-active phospholipids that are secreted by epithelial cells. These phospholipids may protect the mucosa by forming a hydrophobic layer that repels acid at the luminal surface of the mucus gel.As a result of these pre-epithelial defense mechanisms, the pH on the surface of the gastroduodenal epithelial cell normally can be maintained in the neutral range even when pH in the lumen falls below 2.8. finally, acid-peptic injury to the gastroduodenal mucosa results in an outpouring of mucus, fibrin and cellular debris that form a protective cap which clings to the injured epithelium and impedes further contact with acid.

Abnormalities in these pre-epithelial defense mechanisms may contribute to peptic ulcer disease. For example, H.pylori infection can be associated with abnormalities in gastrointestinal mucus and in duodenal bicarbonate secretion that predispose to peptic ulceration.

EPITHELIAL DEFENSE MECHANISMS:

When acid and pepsin breach the pre-epithelial defenses, epithelial mechanisms can prevent or minimize acid-peptic injury. The apical cell membranes and the tight junctional complexes between the surface cells are barriers that limit the diffusion of hydrogen ions into the mucosa.

Exposure of the apical cell membranes to dilute acid causes an increase in resistance to the passage of hydrogen ions through the tight junctions, whereas exposure to more concentrated acid (pH<2.5) induces injury that allows hydrogen ions to leak through this paracellular pathway[6]. Excess hydrogen ions that enter the epithelial cells can be removed by ion pumps in the basolateral cell membrane that include a Na^+/H^+ exchanger and a CI^-/HCO_{-3} exchanger.

Duodenal epithelial cells also have a Na/HCO_3 cotransporter that helps to regulate intracellular pH. When these defense mechanisms are overwhelmed and cells succumb to acid-peptic injury, superficial mucosal defects can be sealed quickly through a process called rapid restitution in which healthy cells in the mucus neck region of the gland migrate along the basement membrane to close the mucosal gap. This process is regulated in part by growth factors such as epidermal growth factor and fibroblast growth factor.

Rapid restitution merely involves cell migration, not cell division, and the wandering cells can seal only minor mucosal defects. The healing of large peptic lesions is effected through regeneration, a process in which new cells are created by cell division. Regeneration is also regulated by growth factors.

POST-EPITHELIAL DEFENSE MECHANISMS:

Mucosal blood flow comprises the post-epithelial defense mechanism. Blood flow provides much of the energy and the substrates necessary both for maintaining epithelial cell integrity and for effecting protective epithelial cell functions such as mucus production and bicarbonate secretion. Blood flow also removes acid that diffuses through an injured mucosa.

During gastric acid secretion, HCO_3 transported across the parietal cell basolateral membrane produces an “alkaline tide” in the sub mucosa. Blood flow transports the HCO_3 of this alkaline tide to the surface epithelial cells, a process that appears to protect against acid-peptic injury during acid secretion by the stomach.

Peptic ulceration results when the caustic effects of acid and pepsin in the gastrointestinal lumen overwhelm all three components of epithelial defense.

NSAID AND PEPTIC ULCER:

The large majority of peptic ulcerations that are not associated with H. pylori infection are associated with NSAID ingestion. Peptic ulceration with NSAIDs typically cause no symptoms, but NSAID induced ulcers can be symptomatic and complicated by GI bleeding, perforation, and/or obstruction.

Superficial gastric lesions such as petechiae and erosions are found in approximately 50% of individuals who chronically consume NSAIDs, but these lesions appear to have little clinical importance. Asymptomatic ulcerations can be documented endoscopically in 15% to 45% of patients on chronic NSAID therapy. However, 1% to 4% of patients receiving NSAIDs for 1 year will experience serious GI complications[7].

PATHOPHYSIOLOGY OF NSAID ULCERS:

The pathophysiology of NSAID-induced injury can be grouped into two categories: those dependent on inhibition of the enzyme cyclooxygenase and those independent of cyclooxygenase inhibition. The later category comprises local mucosal toxic processes.

Topical effects of NSAIDs are likely the major mechanism responsible for the acute hemorrhages and erosions observed acutely after NSAID challenge. Within a few minutes of NSAID ingestion, denudation of surface epithelial cells and increased mucosal permeability occur.

Most NSAIDs are weak organic acids that, in acidic gastric juice, are un-ionized and thus freely lipid soluble. The lipid-soluble, un-ionized NSAIDs diffuse across gastric mucosal epithelial cell membranes into the cytoplasm, where they ionize at neutral pH and thus become “trapped” within the cells.

The high intracellular concentrations of NSAIDs cause local toxic effects. One mechanism of these local effects is an uncoupling of oxidative phosphorylation, resulting in decreased mitochondrial energy production, a reduction in cellular integrity and increases in cellular permeability.

Another topical mechanism of NSAID injury is an attenuation of the phospholipid content and surface hydrophobicity of the gastric mucus gel layer. Some NSAID metabolites that are excreted in bile can also cause topical injury to the gastrointestinal mucosa.

The most important risk factor for an NSAID-induced complication is a history of prior peptic ulcer disease or a prior ulcer complication factors that increase the risk for NSAID-induced GI events by twofold to fourfold. Advanced age is also a substantial risk factor. Although there also appears to be a threshold age at which risk dramatically increases, the relative risk increases linearly at the rate of approximately 4% per year of advanced age. Data on the role that duration of NSAID exposure has in the risk for GI events have been conflicting.

Some case-control studies have suggested that the risk of NSAID-associated gastrointestinal complications is highest within the first 30 days of NSAID use.

It has become clear from epidemiologic studies that as the dose of an NSAID increases, the risk of ulcer complications also increases in parallel fashion.

Other risk factors are concomitant use of flucocorticoids or anticoagulants and comorbid conditions such as significant heart disease or rheumatoid arthritis.

NSAID use and H.pylori infection generally have been regarded as independent risk factors for peptic ulcer disease[8]. However, evidence is accumulating that H.Pylori infection and NSAID use may be more than just additional risk factors for ulcer disease. NSAID users infected with H.pylori have an almost twofold increased risk for developing bleeding peptic ulcers compared to that with uninfected NSAID users and low dose aspirin causes more gastric injury in H.pylori infected subjects than uninfected individuals.

CIGARETTE SMOKING:

Cigarette smoking is a risk factor for peptic ulcer disease and its complications[9]. Furthermore cigarette smoking may also affect the healing of peptic ulceration and in the absence of treatment for H.pylori, may even predispose to relapse.

Smokers have been found to have decreased prostaglandin concentrations in their gastric and duodenal mucosa, and smokers have shown to inhibit acid stimulated duodenal mucosal bicarbonate secretions.

ALCOHOL INTAKE:

It is a common misconception among clinicians that alcohol ingestion is a strong risk factor for peptic ulcer disease,

The prevalence of ulcer disease appears to be increased for patients with alcoholic cirrhosis but no such association has been established for drinkers without cirrhosis[10], indeed in one the retrospective study suggested that modest alcohol consumption might even protect against peptic ulceration[11].

DIET:

No study has established convincing link between diet and peptic ulcer disease. Ulcer patients often describe dyspepsia associated with ingestion of certain foods mostly spicy foods, but the evidence of such foods causing ulceration is virtually nonexistent, coffee, tea and colas are potent gastric acid secretagogues[12], but epidemiologic studies have not established an association between these beverages and peptic ulcer disease. Of note, both caffeinated and decaffeinated coffee appear to be equal in their ability to stimulate gastric acid secretion[12].

DISEASE ASSOCIATED WITH PEPTIC ULCER:

A number of chronic illness have been associated with peptic ulcer disease. For example, peptic ulcerations have been found in upto 30% of patients with

chronic pulmonary disease[13]. The mechanisms responsible for this association are not clear, although cigarette smoking may underlie both conditions.

Patients with cirrhosis appear to have an increased risk of developing peptic ulceration and its complications[14]. Chronic renal failure has been proposed as a risk factor for a peptic ulcer disease, but studies on this issue are contradictory. Other disorders allegedly associated with peptic ulceration, but for which firm evidence is lacking, include cushings disease, hyperparathyroidism and coronary artery disease. The frequency of H.pylori infection among patients with these chronic disorder is no yetr clear, and the possible contribution of H.pylori to the association of these conditions with peptic ulceration has not yet been explored adequately.

EMOTIONAL STRESS;

Since the recognition of the importance of H.pylori in the pathogenesis of peptic ulcer, however physician interest in the association between emotional stress and ulcer disease has wanted. Emotional stress alone does not appear to be sufficient to cause ulcers in most patients because eradication of H.pylori and elimination of NSAIDS generally prevents ulcer recurrence irrespective of emotional factors. Nevertheless, some modern studies still suggest that stress contributes to peptic ulcer disease[15].

Furthermore, it is not known why only a minority of individuals who take NSAIDS or who are infected with H.pylori develop peptic ulcers, and emotional stress and/or a genetic predisposition may well be risk factor is in these susceptible subjects.

GENETICS

A number of observations have suggested that genetic factors predispose one to the development of ulcer disease.

The genes responsible for this apparent ulcer predisposition are not known. Furthermore, it now appears that some of this familial clustering of ulcer disease is the result of a high rate of H.pylori infection in family members rather than a consequence of genetic factors predisposing to ulcer disease per se. An elevated level of serum pepsinogen-I, initially thought to be a genetic marker for ulcer disease, also appears to be a reversible consequence of H.pylori infection[16]. Other proposed genetic markers for ulcer disease include blood group O antigen, the lack of secretion of blood group antigens in the saliva, and the presence of certain HLA subtype.

The association of certain blood group antigens with peptic ulcer disease may be explicable, at least in part, by the fact that these antigens may affect an individual’s susceptibility to H.pylori infection. For example, Lewis blood group

antigens have been reported to mediate H.pylori attachment to the human gastric mucosa[17]. In the large study of Danish men, Hein and colleagues found that Lewis phenotype Le(a+b) and the ABH nonsector trait, were makers for ulcer disease. The investigators suggested that these traits might confer a genetic susceptibility to H.pylori infection rather than a specific susceptibility to peptic ulceration.

PATHOGENESIS OF DUODENAL ULCER PERFORATION:

Perforation of an ulcer is due to sudden sloughing of base of the ulcer due to impairment of blood supply, which leads to gastric or duodenal contents leaking into the peritoneal cavity, intiating an acute diffuse peritonitis.

Although the initial peritonitis following perforation of peptic ulcer is a chemical one, there is mainly concurrent bacterial contamination which can aggravate the inflammatory process and progress to the development of intra abdominal abscesses over the ensuring days or weeks.

The most serious complications of acute peritonitis are paralytic ileus and toxemia.

Absorption of bacterial endotoxins through the inflamed peritoneum causes endotoxemia, which may result in septicemia. The combination of endotoxic shock and fluid & electrolyte imbalance is the cause for high mortality rate in untreated acute bacterial peritonitis.

Up to one third of patients of duodenal ulcers both the basal acid output and maximal acid output are increased. Those individuals with basal acid output more than 15mEq/h are at high risk. it indicates the increased parietal cell mass

In addition to the increased gastric and duodenal secretions, accelerated gastric emptying is also seen in patients, this leads to increased acid load being delivered to the 1st part of the duodenum which is the most common area for the duodenal

ulcer, acidification inturn leads to gastric metaplasia indicating replacement of duodenal villous cells by secretory gastric epithelium. It further enhances the colonization by H.pylori by creating an environment well suited for the H.pylori.

ACID SECRETION AND PEPTIC ULCER:

A variety of abnormalities related to mucosal acid exposure have been described in patients with duodenal ulcer.

Although duodenal ulcer patients group have a higher mean BAO and mean MAO compared to normal controls but many duodenal ulcer patients have basal and peak acid outputs in the normal range, and there is no correlation between acid secretion and severity of the ulcer disease.

As a group duodenal ulcer patients produce more acid than normal controls in response to any known acid secretory stimulus. Although they usually have normal fasting serum gastrin level, duodenal ulcer patients often produce more gastric acid At any given dose of gastrin than controls.

Considering that many duodenal ulcer patients do produce excessive gastric acid, it has been argued that a normal fasting level in these patients is inappropriately high, and that there is an impaired feedback mechanism, especially in light of the apparently increased sensitivity of the parietal cell mass to gastrin. Many of these long standing observations now seem reasonable in view of recently

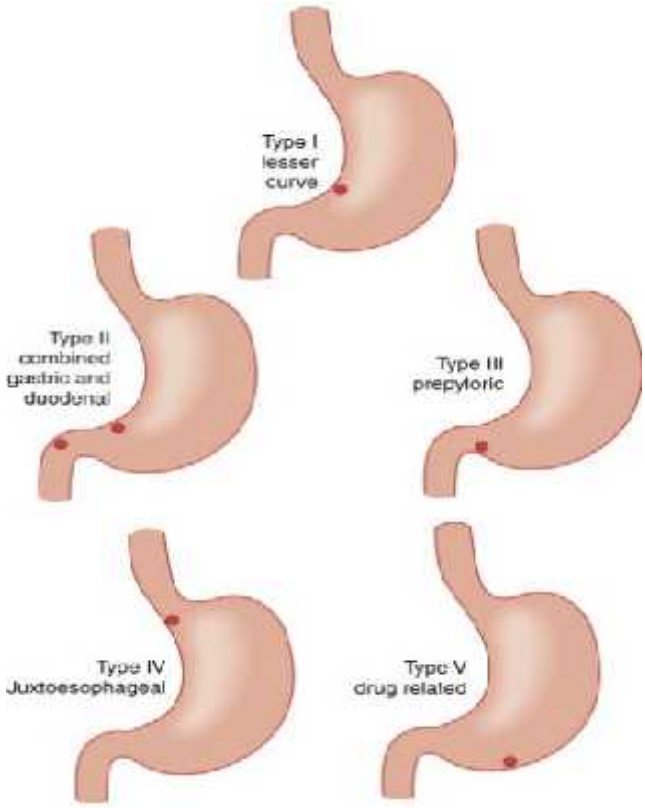
gained understanding of the perturbations in acid and gastrin secretions associated with H.Pylori infections.

Some patients with duodenal ulcer also have increased rates of gastric emptying that deliver and increase acid load per unit of time to the duodenum. Finally, the buffering capacity of duodenum in many patients with duodenal ulcer is compromised due to decreased duodenal bicarbonate secretion. In patients with gastric ulcer, gastric secretion is variable.

Currently five types of gastric ulcer are described, although the original Johnson's classification contained three types. The most common,

- Type 1 gastric ulcer,is typically located near the Angularis incisura on the lesser curvature, close to the border between the antral and corpus mucosa. Patients with type 1 gastric ulcer usually have normal or decreased acid secretion.
- Type 2 gastric ulcer is associated with active or quiescent duodenal ulcer disease, and
- Type 3 gastric ulcer is prepyloric ulcer disease. Both type 2 and type 3 gastric ulcers are associated with normal or increased gastric acid secretion.

- Type 4 gastric ulcers near the GE junction, acid secretion is normal or below normal.



- Type 5 gastric ulcers are medication induced and may occur anywhere in the stomach.

Patients with gastric ulcers may have weak mucosal defenses that permit an abnormal amount of injurious acid back diffusion into the mucosa.

Duodeno-gastric reflux may play a role weakening the gastric mucosal defenses, and a variety of components in duodenal juice, including bile, lysolecithin, and pancreatic juice, have been shown to cause injury and inflammation in the gastric mucosa. NSAIDS and aspirin have similar effects.

Other chronic gastric ulcer usually is associated with surrounding gastritis, it is unproven that the later leads to the former.

OPERATION FOR DUODENAL ULCER:

BILLROTH II GASTRECTOMY:

The first successful gastrectomy was performed by Billroth in January 1881, Wolfler performed the first gastro enterostomy in the same year. The original Billroth operation consists of a gastric resection with gastro duodenal anastomosis (Billroth I technique).

The Billroth II operation was devised more by accident than design. A gastro enterostomy was performed on a gravely ill patient with a pyloric cancer, who was not expected to survive. Contrary to expectations, the patient improved and the stomach distal to the anastomosis was resected.

It soon became evident that the use of a gastrojejunal anastomosis after gastric resection could be safer and easier than Billroth I procedure, and it became popular and effective in the surgical treatment of duodenal ulcer.

Because of its disadvantages such as higher operative mortality and morbidity, it has not been used for many years in the patient with uncomplicated ulcers, but it is still used occasionally in the treatment of a complicated ulcer with a

difficult duodenum. In Billroth II gastrectomy, or its close relation polya gastrectomy, the antrum and distal body of stomach are mobilized by opening the greater and lesser omentum and dividing the gastro epiploic arteries, right gastric artery and left gastric artery arcade at the limit of the resection. The duodenum is closed off either by suture or using staplers, sometimes with difficulty in patients with a very deformed duodenum.

Various techniques are available to close the difficult duodenum , a catheter may be placed in the duodenal stump, the duodenum closed around it and a catheter brought out through the abdominal wall. Following resection, the distal end of the stomach is narrowed by the closure of the lesser curve aspect of the remnant. The greater curve aspect is then anastomosed, usually in a retrocolic fashion, to the jejunum leaving as short an afferent loop as feasible.

Even when well performed, this procedure has an operative mortality rate of a few per cent and morbidity is not unusual. A common cause of morbidity is leakage from the duodenal stump, which is particularly associated with kinking of the afferent loop. Leakage from the gastrojejunal anastomosis is unusual unless it is under tension or the stomach has been devascularised during the mobilisation.

The incidence of side effects following gastrectomy is considerable. Recurrence of the ulcer at the stoma is uncommon but can occur, especially as this procedure is traditionally not combined with the vagotomy.

GASTROJEJUNOSTOMY:

Because of the potential for mortality after gastrectomy, the use of gastrojejunostomy alone in the treatment of duodenal ulceration was developed. Reflux of alkali from the small bowel into the stomach reduced duodenal acid exposure and was often successful in healing the ulcer.

However, because the jejunal loop was exposed directly to gastric acid, stomal ulceration was extremely common, hence the procedure in isolation was ineffective.

TRUNCAL VAGOTOMY AND DRAINAGE:

Truncal vagotomy was first introduced in 1943 by Dragstedt and, for many years, combined with drainage, was the mainstay of treatment of duodenal ulceration. The principle of the operation is that section of the vagus nerves, which are critically involved in the secretion of gastric acid, reduces the maximal acid output by approximately 50 per cent.

Because the vagal nerves are motor to the stomach, denervation of the antropyloro duodenal segment results in gastric stasis in a sub-Lester Reynolds Dragstedt , 1893–1975, Professor of Surgery, Chicago, IL, USA. He was a physiologist to start with and later became a surgeon. His physiological mind made him consider that vagotomy would be a treatment for peptic ulcer.

Accordingly, he performed the first vagotomy in 1943.stantial proportion of patients on whom truncal vagotomy alone is performed. This was first noted by Dragstedt, who did not perform a drainage procedure when he first introduced the operation.

The most popular drainage procedure is the Heineke–Mikulicz pyloroplasty . It is simple to perform and involves the longitudinal section of the pyloric ring. The incision is closed transversely. Gastrojejunostomy was the alternative drainage procedure to pyloroplasty. This is performed through opening the lesser sac and performing an anastomosis between the most dependent part of the antrum and the first jejunal loop. An isoperistaltic anastomosis was most commonly performed.

The operation of truncal vagotomy and drainage is substantially safer than gastrectomy. However, the side effects of surgery are, in fact, little different from those that follow gastrectomy.

HIGHLY SELECTIVE VAGOTOMY:

In 1968, Johnston and Amdrup independently devised the operation of highly selective vagotomy in which only the parietal cell mass of the stomach was denervated.

This proved to be the most satisfactory operation for duodenal ulceration, with a low incidence of side effects and acceptable recurrence rates when performed to a high technical standard. This operation became the ‘gold standard’ for operations on duodenal ulceration in the 1970s.

The operative mortality was lower than any other definitive operation for duodenal ulceration, in all probability because the gastrointestinal tract was not opened during this procedure. The unpleasant effects of peptic ulcer surgery were largely avoided, although loss of receptive relaxation of the stomach did occur, leading to epigastric fullness and sometimes mild dumping. However, the severe symptoms that occur after other more destructive gastric operations did not occur.

It is often said that recurrent ulceration is the Achilles heel of this operation although, when performed well, recurrence was no more common than after truncal vagotomy. The operation disappeared from routine use with the advent of antisecretory agents and eradication therapy.

TRUNCAL VAGOTOMY AND ANTRECTOMY:

For completeness, this operation should be mentioned as at one stage it was popular in the USA. In addition to a truncal vagotomy, the antrum of the stomach is removed, thus removing the source of gastrin, and the gastric remnant is joined to the duodenum.

The recurrence rates after this procedure are exceedingly low. However, the operative mortality is higher than after vagotomy and drainage and the incidence of unpleasant side effects is similar.

OPERATIONS FOR GASTRIC ULCER:

In contrast with duodenal ulcer surgery, when the principal objective was to reduce duodenal acid exposure, in gastric ulceration the diseased tissue is usually removed as well.

This has the advantage that malignancy can then be completely excluded. As with duodenal ulceration, such surgery is not now performed except for complications of gastric ulcer.

BILLROTH I GASTRECTOMY:

This was the standard operation for gastric ulceration until Medical Treatments became prevalent. The distal stomach is mobilised and resected in the

same way as in the Billroth II gastrectomy. This resection should include the ulcer that is usually situated on the lesser curve. The cut edge of the remnant is then partially closed from the lesser curve aspect, leaving a stoma at the greater curve aspect, which should be similar in size to the duodenum. Reconstruction may be facilitated by mobilising the duodenum using Kocher's manoeuvre.

The incidence of recurrent ulceration after this operation is low, but it carries with it the morbidity and mortality associated with any gastric resection.

BACTERIOLOGY

Most common organism isolated from peritoneal fluid are E.coli, staphylococci, streptococci and anaerobic organism[18].

ASSOCIATION OF HELICOBACTER PYLORI AND PEPTIC ULCER

H.pylori infections are more common with recurrent peptic ulcer perforation and is present in half of world's population.

It is a microaerophilic, curve, gram negative urease producing bacterium which is able to colonize to surface of the mucosa, deep into the overlying mucus layer.

H.pylori infections is present in virtually all patients with duodenal ulcer and about 70% of the patients with gastric ulcer. Antibiotic treatment of H.pylori infection promotes healing of ulcers and tends to prevent the recurrence[18,21].

H.pylori exerts its pathological effects through the elaboration of enzymes and toxins. The mucosal barrier is disturbed by the endopeptidase which have a powerful mucolytic action.

The epithelial surface is altered by the H.pylori by generation of large amount of ammonia. pH alteration in mucosal charge gradient, epithelial sodium potassium ATPase activity and cellular permeability leads back diffusion of hydrogen ions. Clinically the diagnosis of H.pylori infection is done by chromo endoscopy urease breath test, rapid urease test.

H.pylori fecal antigen test: this test is relied on the monoclonal antibody immune-chromotography of the fecal sample. It is both specific and sensitive and can detect the organism in its early stage of the disease.

Urea breath test: labeled with a carbon isotope (carbon 13 or carbon 14). After a few minutes, the concentration of the labeled carbon is measured in breath. The concentration will be high only when urease is present in the stomach.

Because the human stomach does not produce urease, such a reaction is possible only with H. *pylori* infection

H pylori serology

This serological test has a high (>90%) specificity and sensitivity. It is currently based on the quantitation of IgG antibodies against *H.pylori* by the means of an enzyme-linked immunosorbent assay.

It is useful for detecting a new cases, but cannot be used for follow-up of treated patients because the results do not indicate present infection with *H. pylori*. The antibody titer may remain elevated for a long time after H.pylori eradication. The number of false-positive results is related to age and increases with age.

The organism can be detected microscopically using Giemsa and warthin starry silver stain.

The medical management of H.pyori infection is achieved by triple therapy which includes, omeperazole, amoxicillin and clarithromycin for 14 days or bismuth subsalicylate,metronidazole and tetracycline or lansoperazole, amoxicillin and clarithromycin. Now there is trend of antibiotic resistance leading to increase in failure rates of this standard regime

CLINICAL FEATURES

Perforation are classified into

Acute perforation

Slow perforation

Sub acute perforation

Chronic perforation

ACUTE PERFORATION:

Clinical course of perforation can be divided into three stages

Primary stage or stage of peritoneal irritation

Secondary stage or stage of peritoneal reaction

Tertiary stage or stage of bacterial peritonitis

PRIMARY STAGE:

This stage occurs in the initial 2 to 3 hours of perforation. The patient will be pale and anxious. The patient may exhibit pain, sweating, difficulty in respiration and peripheral vasoconstriction.

The patient will have tachycardia and the temperature maybe subnormal. On examination the abdomen may move a little or does not move with respiration, diffuse tenderness may be present and may present with board like rigidity. Abdomen will be dull on percussion and pelvic tenderness may be elicited on per rectal examination.

SECONDARY STAGE

This stage last from 3 to 6 hours. It depends on the defense mechanism. There may be diffuse chemical peritonitis or sealing of perforation may occur. The general condition of the patient improves apparently.

Pain, tenderness and rigidity may get reduced. Temperature is normal and the pulse rate raises still higher and the bowel sounds are absent. This is known as period of illusion as there is a temporary improvement of general condition of the patient.

TERTIARY STAGE:

This stage occurs after six hours with severe peritonitis. There will be silent abdominal distension and there will be enough free fluid in the abdomen that can be clinically detectable. The pulse rate becomes higher which marks the detoriaration of the patients general condition.

SLOW PERFORATION:

Pain may be less severe, less generalized with equivocal guarding and rigidity. Bowel sound may be present and a small amount of free fluid may track down the right paracolic gutter causing pain and tenderness in the right iliac fossa which simulates appendicitis.

SUBACUTE OR FORMED FRUSTA

Ulcer may perforate and sealed rapidly, before contamination of general peritoneal cavity. Guarding, rigidity in epigastrium and right hypochondrium. Abdomen soft. This type of presentation is known as formed frusta.

CHRONIC OR CONFINED PERFORATION

When full thickness hole is created by an ulcer but free spillage is prevented by contiguous organ such as colon, greater omentum creating a walled off area. Signs and symptoms are absent.

PERFORATION AND HAEMORRHAGE

The combination of haemorrhage and perforation occurs in 3 ways.

- ❖ Concomitant haemorrhage and perforation
- ❖ Perforation during medical treatment of haemorrhage
- ❖ Haemorrhage after a recently sutured perforation.

INVESTIGATION

Appropriate laboratory evaluation of patients with suspected perforation includes a complete blood count, serum electrolytes and amylase.

Patients presenting late following perforation may require assessment of renal function with a serum creatinine and pulmonary function and acid base balance with an arterial blood gas. Leukocytosis with a left shift is usually present, but may be absent in the immunosuppressed or elderly patient. Serum amylase is most commonly normal, but elevated levels less than three times normal are occasionally encountered. Liver function tests are usually normal.

Unless the presentation is delayed, serum electrolytes and renal function are normal.

X RAY

A chest radiograph and spine and abdomen erect and left decubitus abdominal films should be obtained. Free air in the peritoneal cavity is seen in 70% of patients[22].

The absence of free air, therefore, does not exclude the diagnosis. When perforation is suspected, but pneumoperitoneum is absent, an upper gastro intestinal study with water soluble contrast may establish the diagnosis. With clear-cut signs of peritonitis, however, such contrast studies are unnecessary.

CAUSES OF PSEUDOPNEUMO PERITONEUM

- Chiladiti syndrome
- Subdiaphragmatic fat
- Curvilinear pulmonary collapse
- Omental fat
- Subphrenic abscess
- Subpulmonary pneumothorax
- Intramural gas in pneumatosisintestinalis

CONTAST RADIOGRAPHY

In doubtful cases, gastrograffin is used to differentiate sealed from unsealed perforation.

USG/CT SCAN ABDOMEN

It can detect free intraperitoneal air and free fluid in the abdomen

DIFFERENTIAL DIAGNOSIS:

It can be divided into intra abdominal, intra thoracic and metabolic causes.

INTRA ABDOMINAL

- Acute appendicitis
- Acute pancreatitis
- Acute cholecystitis
- Acute intestinal obstruction
- Mesentric vessel disease
- Ruptured ectopic
- Traumatic perforation
- Appendicular perforation
- Meckels diverticultis

INTRA THORACIC

- Pneumonia
- Pleurisy
- Myocardial infarction
- Spontaneous pneumothorax
- Rupture of oesophagus

METABOLIC AND NEUROLOGIC

- Acute porphyria
- Diabetic ketoacidosis
- Uraemia
- Multiple sclerosis
- Neuro syphilis

MANAGEMENT

Management includes insertion of naso-gastric tube and aspiration. correction of hydration, monitoring of urine output, IV antibiotics, combination therapy with ampicillin, gentamycin and metronidazole.

Central haemodynamic monitoring in unstable patients or patients with significant cardiopulmonary risk. The main aim of initial management is to re establish intra vascular fluid volume to decrease the risk of anasthesia induction.

SURGICAL MANAGEMENT

The patient with perforated duodenal ulcer should be ideally operated with an hour of presentation. In any delay in treatment especially more than 24 hours significantly increases the mortality and morbidity rates and prolong the length of hospital stay.

Various management techniques are

Simple closure with live omental patch

Closure with serosal onlay patch

Simple closure with definitive procedure for ulcer

Endoscopic closure of perforated ulcer

Laparoscopic closure of perforated ulcer

Flank drain and conservative management

We commonly do simple closure with live omental patch technique in our institution.

NON OPERATIVE MANAGEMENT

Occasionally patient may present late i.e. More than 24 hours after perforation. Non operative or conservative management are considered in this group of patients. If

The patient is haemodynamically stable

Generalized peritonitis is absent

Water soluble contrast examination showing no free fluid in the peritoneum.

Management of these patients include nasogastric suction, correction of dehydration, intra venous H2 receptor antagonist or proton pump inhibitors administration and broad spectrum intra venous antibiotics and close clinical monitoring.

Surgical management should be immediately considered in these patients if the clinical condition deteriorates. These patients are also susceptible to the consequent development of sub phrenic or sub hepatic abscess.

Previous retrospective studies suggested a more frequent use of non operative management even in cases of early perforation as long as there is no free fluid leak and absence of diffuse peritonitis.

A recent randomized prospective trial from Hongkong also confirms that non operative management is safe in patients who are haemodynamically stable and without diffuse peritonitis[19]. But sincere caution should be exercised if this approach is applied on elderly and frail patients.

Only 1/3rd of the patients more than age of 70 years were managed successfully, non operatively in the Hongkong series compared to those with nearly 80% were managed successfully in the 40 to 70 years age group and in case of less than 40 years of age 100% results were achieved. Old age patients are liable to complications related to the failure of non operative method and early surgical management may be preferable for these patients.

DEFINITIVE PROCEDURES FOR DUODENAL PERFORATION

Truncal vagotomy with suitable drainage procedures

Highly selective vagotomy

INDICATIONS FOR DEFINITE ULCER SURGERY:

Perforation of less than 24 hours duration

Young and haemodynamically stable patients

No associated comorbid illness

History of peptic ulcer disease

Perforation occurred during anti secretory treatment

Previous ulcer complications

CONTRAINDICATIONS

Comorbid illness

Delay in presentation ie. More than 24 hours

Traumatic perforation

POST OPERATIVE MANAGEMENT

H2 receptor antagonist or proton pump inhibitors given for 4 to 6 weeks[20].

Antibiotic therapy for H.pylori infection. Most regimens of H.pylori management includes a proton pump inhibitor which acts by decreasing the acid production and allows the damaged tissue to heal.

POST OPERATIVE COMPLICATIONS

Wound infection

Intra peritoneal abscess

Duodenal fistula

Respiratory complications

DVT and pulmonary embolism

Septicemia

PROGNOSIS

Prognosis depends on

Amount of fluid in the peritoneal cavity

Size of perforation

Elderly age

Associated co-morbid illness

Delay in presentation

MATERIALS AND METHODS

This study comprises of prospective analysis of the patients diagnosed with duodenal ulcer perforation in Tirunelveli Government Medical College, Tirunelveli during the period from November 2015 to May 2017.

The following data were collected from the hospital records; Age,sex, previous history of ulcer, NSAID intake, duration of symptoms, size of perforation and the amount of peritoneal contamination. The outcome of treatment was elaborated by post operative complications, hospital stay and death.

50 cases of duodenal perforation were studied over a period of 18 months .Of these 50 cases 47 undergone laparatomy, the perforation in all these cases were present in the anterior aspect of 1st part of the duodenum. The patients were treated with perforation closure with live omental patch repair after initial resuscitation and correction of electrolyte imbalances under the cover of broad spectrum antibiotics.

All the patients were continued treatment with anti H.pylori regimen postoperatively.

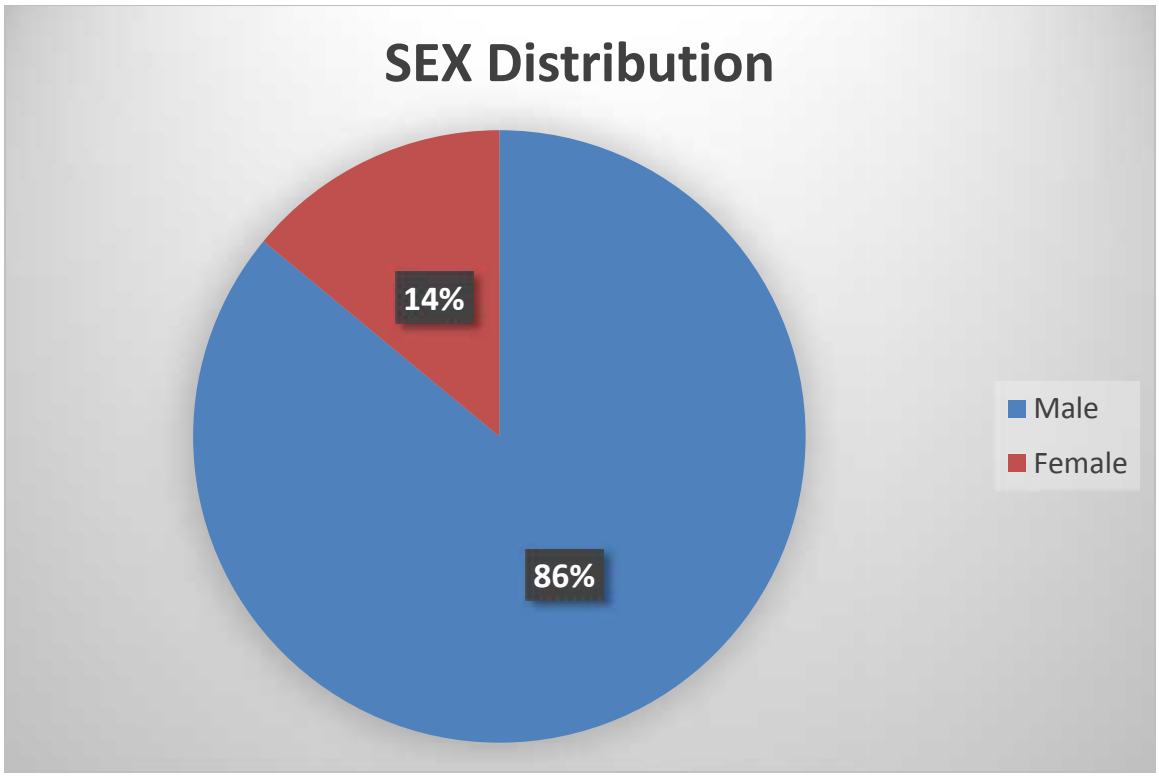
RESULTS

The observations made were

SEX DISTRIBUTION

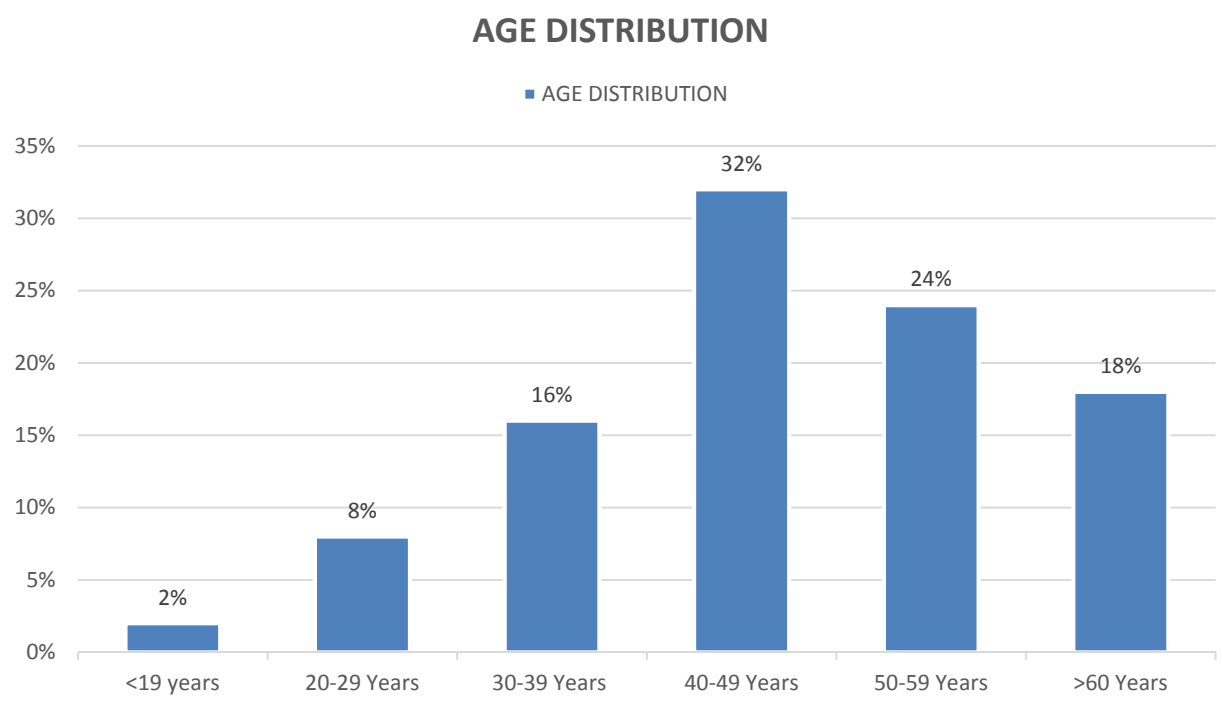
7 female and 43 male patients were noted

Male to female sex ratio is 16.2:1



AGE DISTRIBUTION

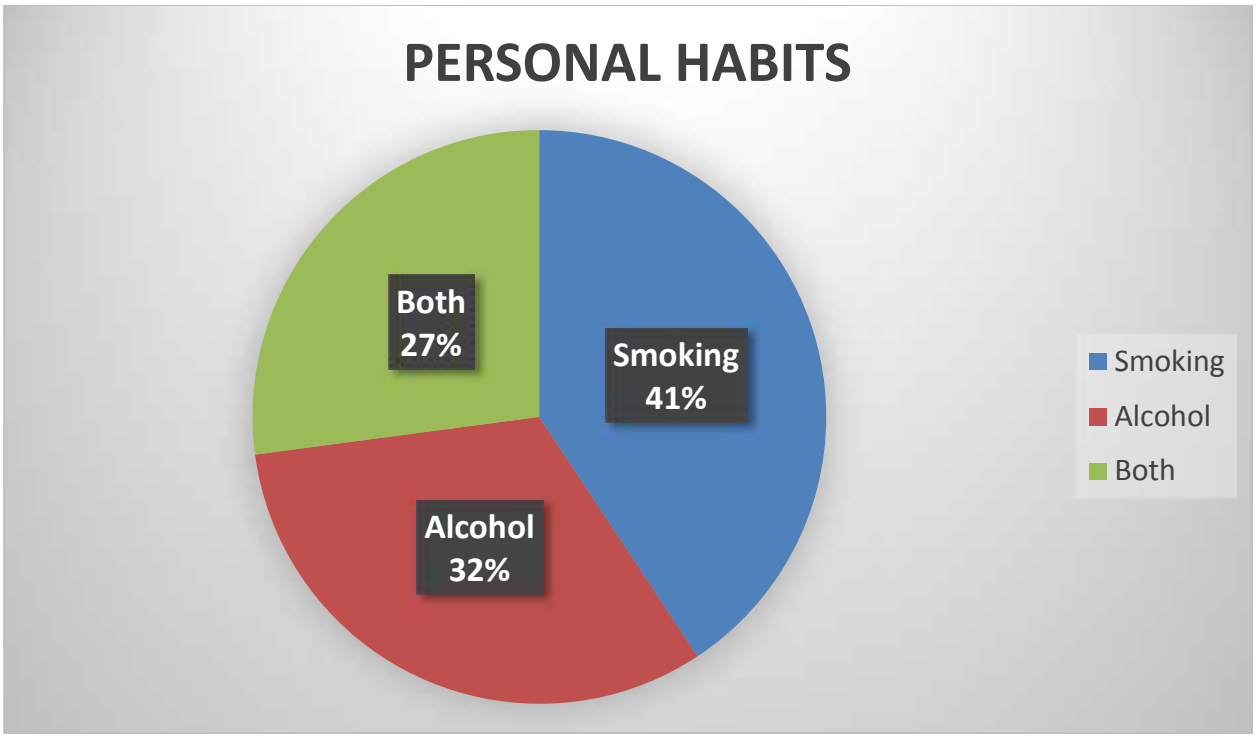
Most of the cases were in the more than 40 years age group and 40 to 50 years is the most frequent numbers in them



AGE	No. of Case	Percentage
<19 years	1	2%
20-29 Years	4	8%
30-39 Years	8	16%
40-49 Years	16	32%
50-59 Years	12	24%
>60 Years	9	18%

PERSONAL HABITS

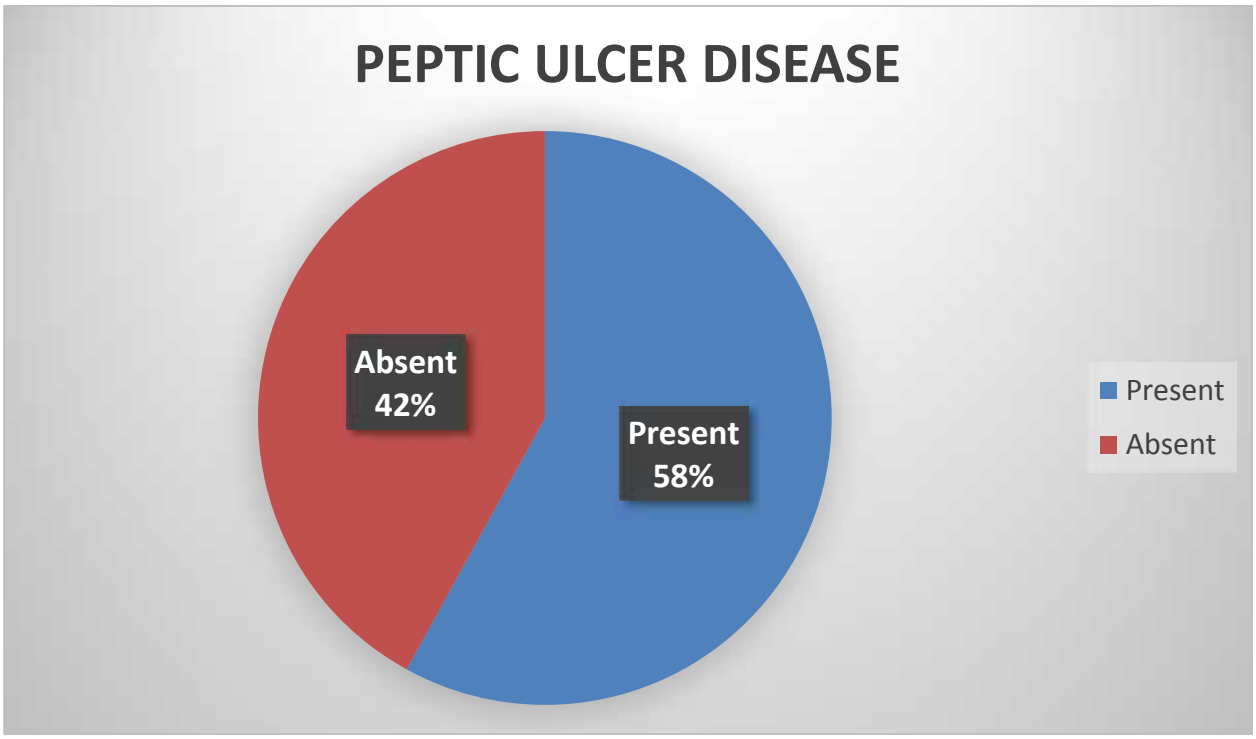
24 patients had smoking history and 19 patients had history of alcohol consumption



Smoking	24	40.7%
Alcohol	19	32.2%
Both	16	27.1%

PEPTIC ULCER DISEASE

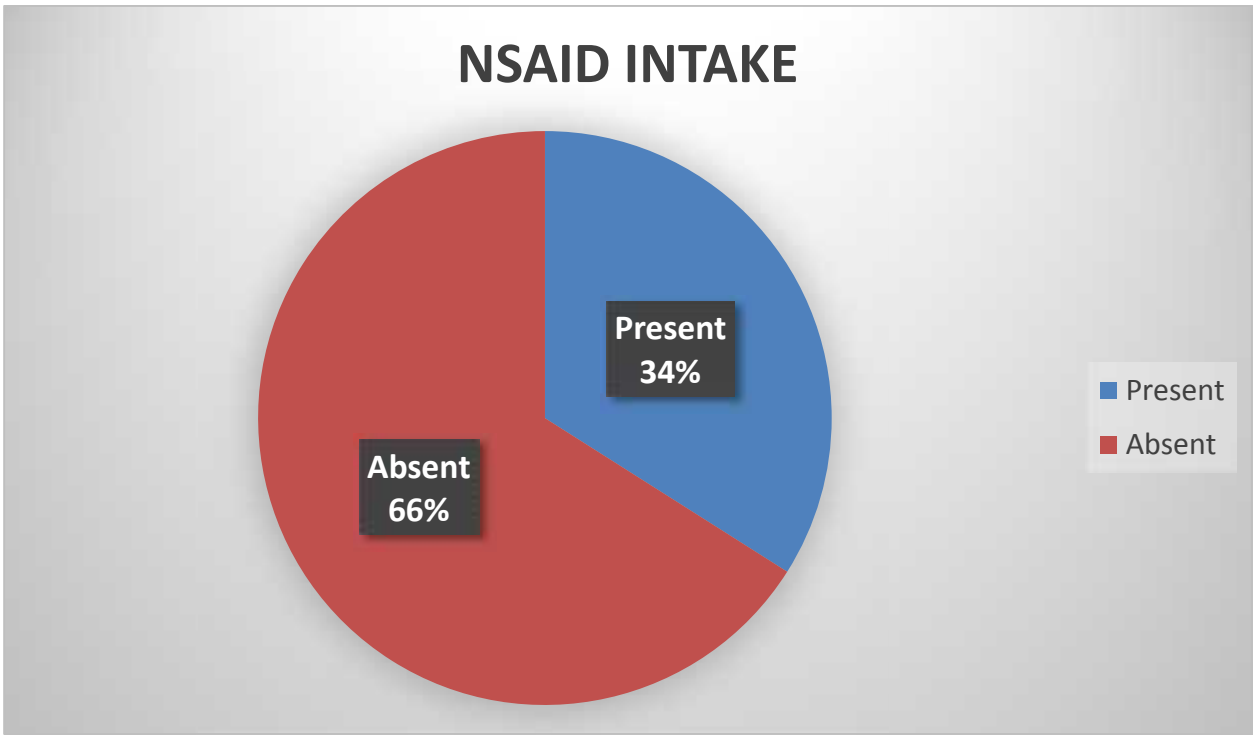
29 patients had the previous history of peptic ulcer disease



Present	29	58%
Absent	21	42%

NSAID INTAKE

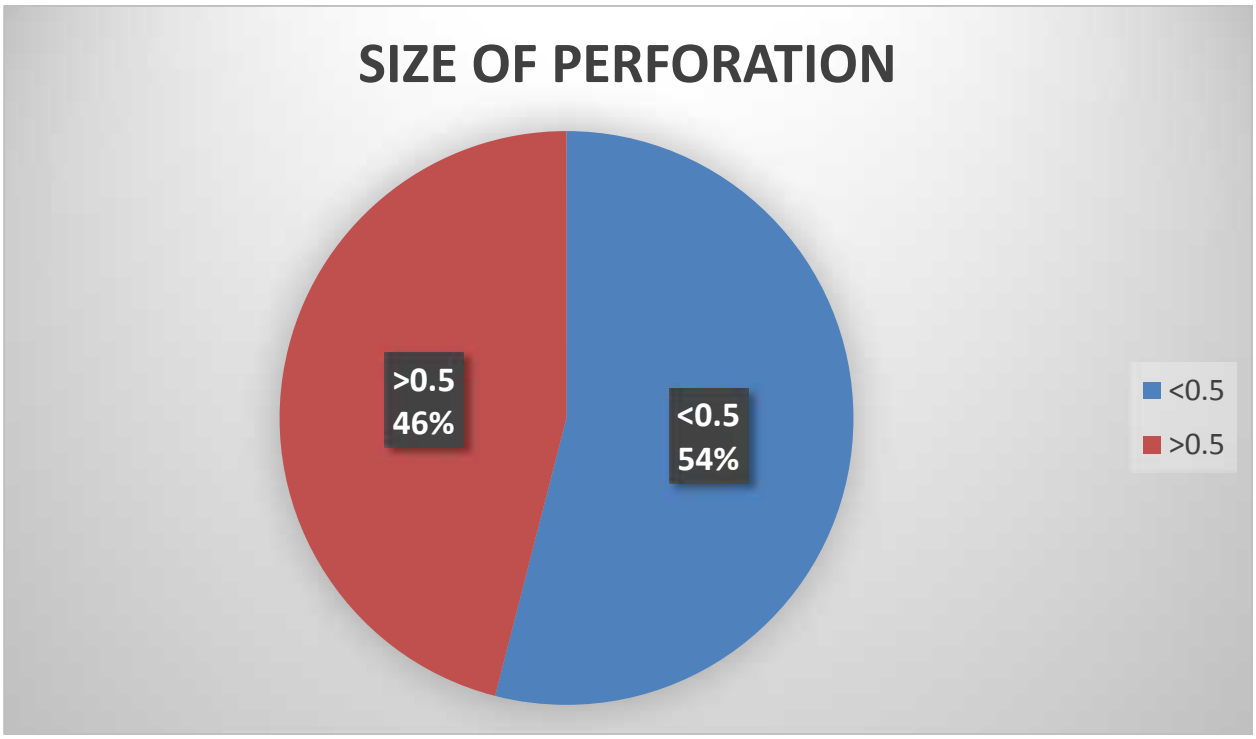
17 patients had the history of NSAIDS intake



Present	17	34%
Absent	33	66%

SIZE OF THE PERFORATION

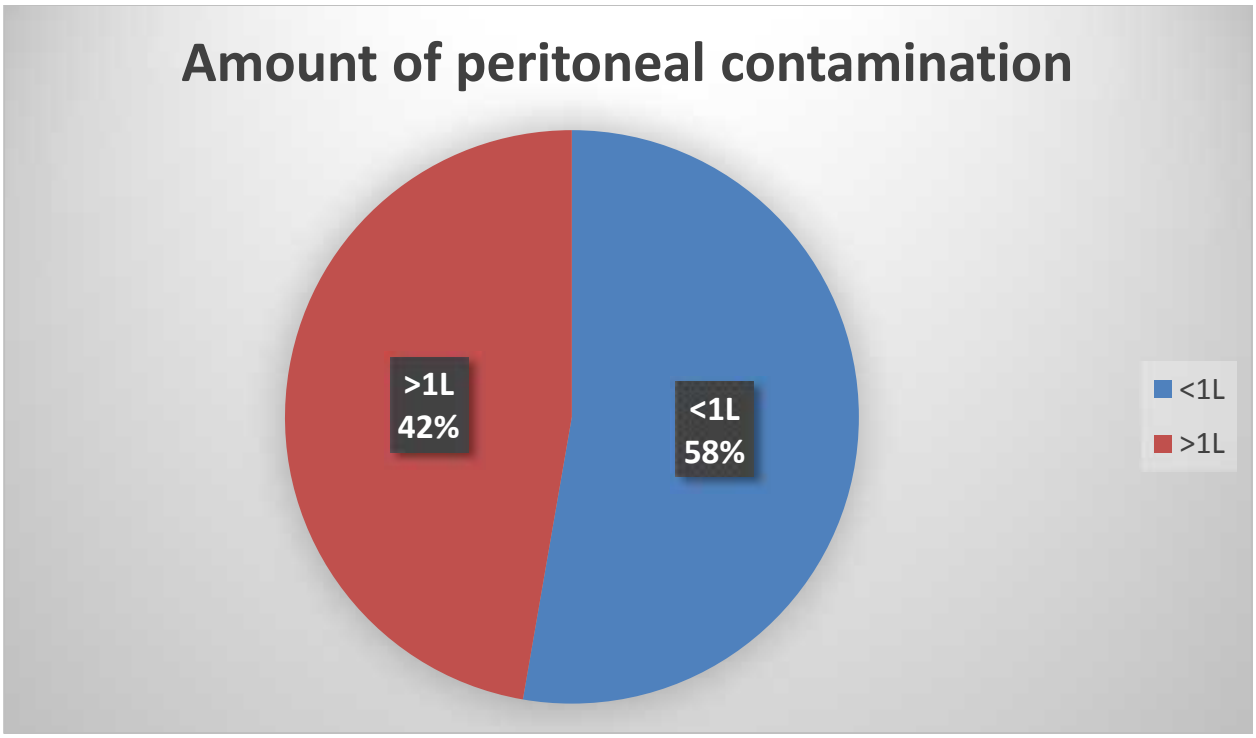
The size of the perforation > 0.5 mm noted in 23 patients



<0.5	27	54%
>0.5	23	46%

AMOUNT OF PERITONEAL CONTAMINATION

The amount of peritoneal contamination more than 1 litre noted in 29 patients. Less than 1 litre noted in 21 patients



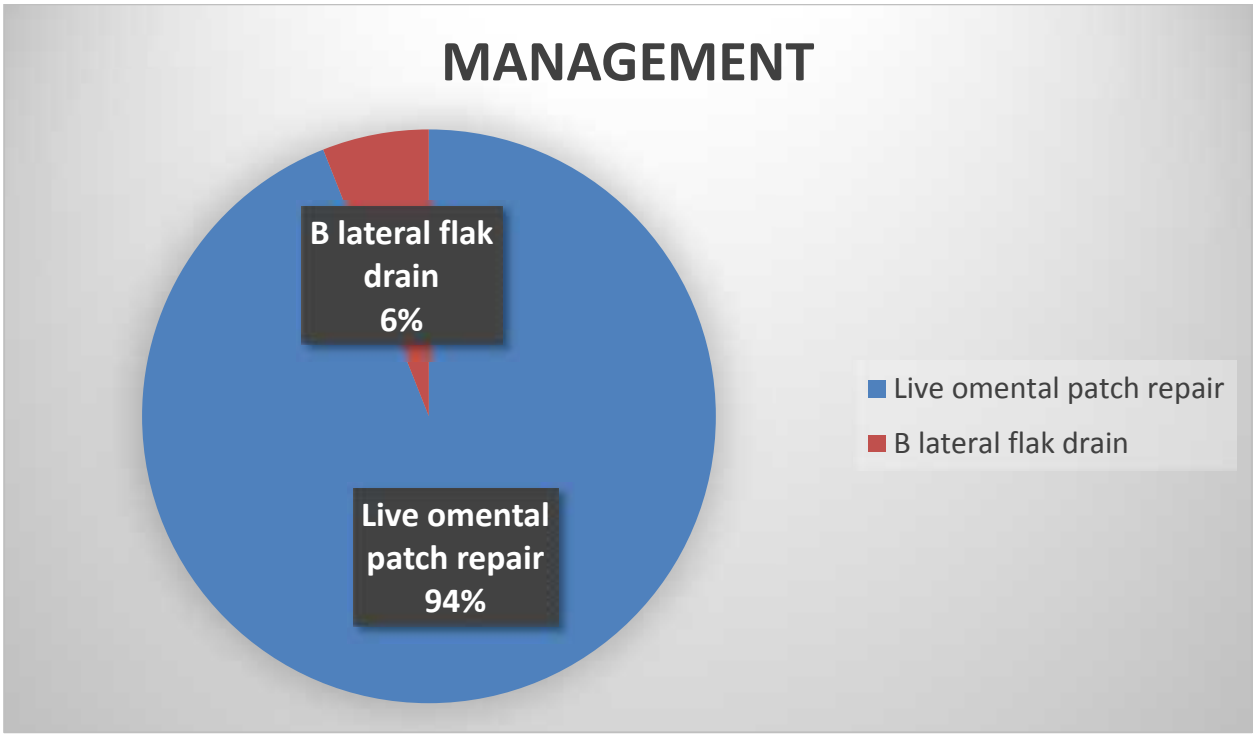
<1L	29	58%
>1L	21	42%

MANAGEMENT

- Out of 50 Patients

- 47 cases were treated with simple closure with omental patch

3 cases were treated with B/L flank drain under local anaesthesia because of poor general condition of the patient



Live omental patch repair	47	94%
Bilateral flak drain	3	6%

DURATION OF SYMPTOMS

- Average duration of symptoms was found to be 1.24 Days (1-4 Days)

DELAY

- Average delay from the time of admission to surgery is 3.48 Hours 2-6Hours)

HOSPITAL STAY

- Average duration of hospital stay found to be 8.52 (8-15 Days)

POST-OPERATIVE COMPLICATIONS

- Of total 47 cases had underwent surgery, 8 Patients had wound infection
- 3 patients had septicaemia
- Electrolyte abnormalities were encountered in 21% of patients
- Morbidity rate: 17.02%.

Mortality

- Of total cases 50, 6 Patients expired due to septicaemia
- Mortality rate:12%

DISCUSSION

AGE DISTRIBUTION

Duodenal ulcer perforation is more common in age group of 40-49 years as per our study

PEAK AGE INCIDENCE	
Debakey et al	>50Years
Taylor et al	>50 Yeats
Hannah et al	31-40 years
Our study	40-49 years

SEX

- Male to female ratio was found to be 16.2:1

Gender incidence	Male to female ratio
Paul H jordan	26:1
Rodney maingot	5:1
Our study	16:1

NSAID INTAKE

- Our study 58%

George stain study	**75 %**
Our study	34%

RADIOLOGICAL SIGNS

- AIR UNDER DIAPHRAGM

Shaffer study	**70%**
Mann et al	85%
Our Study	90 %

HISTORY OF NSAID INTAKE

Patients aged above 50 years with history of NSAID intake are at increased risk for duodenal ulcer perforation, 34% of the patients had history of NSAID intake

The average duration of symptoms was found to be 1.24 days, this is due to most of the patients were managed initially in primary health centres and being referred either delayed.

The average time lag between admission and surgery was found to 3.48 hours, most of the patients needed initial resuscitation initially. In view of improving the general condition of the patient,the patients were made hemodynamically stable and then take up for surgery

POST OPERATIVE COMPLICATIONS

Patients aged more than 60 years and associated comorbid illness had the highest rate of wound infection.

17 patients had associated comorbid illness

Out of the 8 patients who had wound infection 6 patients had associated co morbid illness and 50% of them were above 50 years of age.

Electrolyte imbalance included hyponatremia in 21% of patients and hypokalemia in 19% of patients and elevated serum creatinine in 18% of patients.

Mortality rate was 12% of which 3 patients were treated with B/L flank drain because of the very poor general condition of the patient at the time of admission, and all these patients were above the 60 years age group, of the operated patients 6% mortality is present and in these patients there were associated comorbid illness and delay in presentation and amount of peritoneal contamination were all significantly present .

LIMITATIONS OF THE STUDY:

- A detailed study on the non operative management was not carried out because patients were not randomized. The observations were incidentally made out.
- This study was conducted in tertiary care government setting and may differ from those obtained from primary care setting or private sector.
- Sample size is small
- Therefore the need for further randomized control studies that have a large number of patients and are accurately designed has to be recognized from the present study.

CONCLUSION

- Duodenal ulcer perforation is more common in the age group of > 40 years
- Majority of the patients are male
- Associated risk factors include smoking, alcohol intake, NSAID intake, and history of APD
- Morbidity rate is 17% and wound infection and dyselectolytaemia is the most common
- Mortality rate is 12%
- Mortality and morbidity are significantly higher in patients with comorbid illness.
- age, associated comorbid conditions, duration of symptoms, clinical condition at the time of presentation all contribute in determining the post op morbidity and mortality
- Prognostic indicators can assist in risk stratification for perforated peptic ulcers. The use of this system can help delineate high risk patients and to identify the need of early intervention and prompt treatment for better outcome of the patient.

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PROFORMA

Name: Age: Sex: Ip.No.

Occupation: Rural/Urban:

Date of admission:

Date of surgery:

Date of discharge:

H/o. Pain abdomen

Duration

H/o abdominal distension

H/o APD

H/o drug intake

Previous H/o surgery

Systemic medical illness: Yes/No

Examination

Pulse rate:

BP:

Per abdomen-

Guarding

Rigidity

Distension

Liver dullness obliteration

Bowel sounds

Per rectal examination

Investigations:

Blood urea

Blood sugar

blood Hb:

total count

Differential count

ESR

Serum creatinine

Electrolytes

Chest x ray

X ray abdomen

USG abdomen

ECG

Management:

Time lag

Operative findings: site

Acute or chronic ulcer

Nature of peritoneal fluid

Procedure: simple closure with omental patch/with

definitive surgery

non operative management

Post operative follow up.

Sl. No	IP.No	Name	Age	Sex	Duration in Days	Delay in Hours	Comorbid Illness	H/o APD	H/o. Drug Intake	Size of Perforation >0.5 mm	Amount of Peritoneal contamination >1ltr	Procedure	Anesthesia	Post of Complication	Duration of Stay in Hospital
1	7194	Ramachandran	41	M	2	3	DM	+	+	+	+	SC with OP	GA	Wound Infection	15
2	9065	Mohammed Yosuf	19	M	1	3	-	-	-	-	-	SC with OP	GA	-	10
3	9137	Madathi	36	F	2	4	-	-	-	-	-	SC with OP	GA	-	8
4	9347	Gurusami	46	M	1	3	DM	+	-	+	-	SC with OP	GA	-	8
5	9897	Nagoor	42	M	1	4	HTN		+	-	+	SC with OP	GA	-	8
6	10412	Mariappan	66	M	2		DM	+	-	+	+	B/L Flank Drain	-	Death	1
7	14169	Ganesh	21	M	1	2	-	-	-	-	-	SC with OP	GA	-	8
8	14872	Pachamuthu	44	M	2	3	-	-	-	+	-	SC with OP	GA	-	8
9	15131	Selvam	32	M	1	3	-	+	-	-	-	SC with OP	GA	-	9
10	15781	Suresh	45	M	1	3	-	-	-	+	+	SC with OP	GA	-	8
11	16111	Shammuga Pillai	44	M	1	4	-	+	+	+	-	SC with OP	GA	-	8
12	16543	Thavasimuthu	39	M	1	3	-	-	-	-	+	SC with OP	GA	-	8
13	16892	Muthaiah	67	M	2		-	+	-	+	+	B/L Flank Drain	-	Death	0
14	17017	Subbaiah	49	M	1	3	DM	+	+	+	+	SC with OP	GA	Wound Infection	14
15	18113	Krishnan	54	M	1	4	CAD	-	+	+	-	SC with OP	GA	-	8
16	18744	Vinoth	42	M	1	3	-	+	-	-	+	SC with OP	GA	-	8
17	19021	Mohan	42	M	2	4	DM+HTN	+	+	+	+	SC with OP	GA	Wound Infection	8
18	19314	Saraswathi	26	F	1	3	-	-	-	-	-	SC with OP	GA	-	8
19	19666	Rajesh	64	M	2	4	-	+	-	+	-	SC with OP	GA	-	12
20	19942	Vasantha	43	F	1	3	-	+	-	-	-	SC with OP	GA	-	8
21	20611	Mariappan	53	M	1	4	DM	+	-	+	+	SC with OP	GA	Death 13th POD	-
22	21046	Kannan	45	M	1	3	HTN	+	+	+	-	SC with OP	GA	-	8
23	24631	Chellappa	29	M	1	3	-	-	-	-	-	SC with OP	GA	-	8
24	21998	Kumaran	48	M	1	4	DM	+	+	-	-	SC with OP	GA	-	9
25	32413	Pasupathi	43	M	1	4	-	+	-	+	+	SC with OP	GA	-	8
26	32882	Selvam	23	M	1	3	-	-	-	-	-	SC with OP	GA	-	12
27	33212	Muthukrishnan	34	M	2	4	-	-	-	-	-	SC with OP	GA	-	8
28	33667	Saleem	59	M	1	4	DM+HTN	+	-	-	+	SC with OP	GA	Death 2nd POD	-
29	33921	Rajamani	57	M	1	3	CAD	+	+	+	-	SC with OP	GA	-	8
30	34512	Muthusami	58	M	1	4	HTN	-	+	+	+	SC with OP	GA	-	10
31	39210	Parthiban	46	M	1	4	DM	+	-	-	+	SC with OP	GA	Wound Infection	14
32	41046	Karthikeyan	48	M	1	3	-	-	-	+	-	SC with OP	GA	-	8
33	41824	Arunraja	57	M	1	4	DM	+	-	+	-	SC with OP	GA	-	9
34	42142	Prakash	33	M	2	2	-	-	-	-	-	SC with OP	GA	-	8
35	56210	Gopi	41	M	1	2	-	+	-	+	+	SC with OP	GA	-	8
36	57129	Gomathy	56	F	1	4	DM	+	+	+	-	SC with OP	GA	Death 8th POD	-
37	58584	Thalavai	69	M	2		-	+	+	-	+	B/L Flank Drain	-	Death	0
38	59413	Ponniah	65	M	1	4	-	+	+	-	-	SC with OP	GA	-	10
39	60131	Vellammal	31	F	1	4	-	-	-	-	-	SC with OP	GA	-	8

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Sl. No	IP.No	Name	Age	Sex	Duration in Days	Delay in Hours	Comorbid Illness	H/o APD	H/o. Drug Intake	Size of Perforation >0.5 mm	Amount of Peritoneal contamination >1ltr	Procedure	Anesthesia	Post of Complication	Duration of Stay in Hospital
40	64312	Bala	63	M	1	4	-	+	+	+	+	SC with OP	GA	Wound Infection	14
41	65999	Arumugam	51	M	1	3	DM	+	-	-	+	SC with OP	GA	Wound Infection	14
42	66213	Amuthavalli	53	F	1	4	-	-	-	+	-	SC with OP	GA	-	8
43	66924	Ravi	62	M	2	2	-	-	+	-	+	SC with OP	GA	-	8
44	67210	Gopal	67	M	1	4	-	+	-	-	-	SC with OP	GA	-	8
45	67920	Senthil	52	M	1	4	HTN	-	+	-	+	SC with OP	GA	Wound Infection	14
46	69215	Ganapathy	55	M	1	3	-	+	-	-	-	SC with OP	GA	-	8
47	69960	Sappani	65	M	1	4	-	-	+	-	-	SC with OP	GA	-	8
48	74320	Pappathi	34	F	1	5	-	-	-	-	-	SC with OP	GA	-	8
49	74921	Ravikumar	52	M	2	2	-	+	-	+	+	SC with OP	GA	Wound Infection	15
50	75682	Anthony	38	M	1	4	-	+	-	-	-	SC with OP	GA	-	8